An investigation of functional electrical stimulation cycling for people with spinal cord injury

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Abstract

Spinal Cord Injury (SCI) results in substantial physiological adaptations below the lesion level and reduced cardiopulmonary fitness, resulting in susceptibility to cardiovascular diseases and pressure sores. Functional electrical stimulation (FES) used to produce recumbent cycling has the potential to improve the condition of skeletal muscle, cardiopulmonary fitness and pressure sore risk i.e. health related issues and also enable social and recreational exercise.

Five SCI people (1 female) aged 45.2 ± 3.4 years (mean ± SEM), complete SCI <T3 for at least two years carried out a one year intense FES cycling training programme. Muscle size, strength and contractile properties, peak power output (PO), cardiopulmonary response to exercise and indicators of tissue viability were assessed at three monthly intervals. These data were compared with that from able-bodied (AB) people.

Muscle size and quadriceps strength and fatigue resistance increased significantly and progressively ($P<0.05$) with training but strength remained significantly less than normal ($P<0.01$). Peak PO and the cardiopulmonary response to exercise also improved significantly and progressively ($P<0.05$). There were no significant changes in tissue oxygenation or peak seating pressures however methodological limitations were identified.

Despite these changes the subjects remained highly fatigable during cycling and cardiopulmonary responses indicated high levels of anaerobic metabolism. The increase in PO was less than expected by the increase in isometric strength and generally remained insufficient for outdoor recreational cycling. This may be due to inefficient activation patterns during FES cycling as they were substantially different than those by AB people during voluntary cycling.

FES cycling is a suitable and appealing way to improve health, physical function and social interactions in SCI people and the factors currently limiting the generation of power require further investigation.

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Abbreviations

AB	= Able-Bodied
ACTH	= Adrenocorticotropin Hormone
ATP	= Adenosine Triphosphate
ATPS	= Ambient Temperature and Pressure in Saturated Air
BF	= Breathing Frequency
BFem	= Biceps Femoris
BL	= Blood Lactate
BMI	= Body Mass Index
BDC	= Bottom Dead Centre
BP	= Blood Pressure
bpm	= Beats Per Minute
°C	= Degrees Centigrade
Ca^{2+}	= Calcium
CET	= Constant Load Exercise Test
CFT	= Constant Frequency Train
CHO	= Carbohydrate
cm	= Centimetre
CO_2	= Carbon Dioxide
CSA	= Cross Sectional Area
CV	= Coefficient of Variation
ECG	= Electrocardiogram
End_p	= End of Passive
End_r	= End of Rest
ES	= Electrical Stimulation
g	= Grams
F_ECO_2	= Fraction of Expired Carbon Dioxide
F_EO_2	= Fraction of Expired Oxygen
FES	= Functional Electrical Stimulation
FFA	= Free Fatty Acids
F_ICO_2	= Fraction of Inspired Carbon Dioxide
F_IO_2	= Fraction of Inspired Oxygen
GastL	= Gastrocnemius Lateralis
GastM	= Gastrocnemius Medialis
GH	= Growth Hormone
GMax	= Gluteus Maximus
GMed	= Gluteus Medius
H^+	= Hydrogen ions
HFF	= High Frequency Fatigue
HR	= Heart Rate
HR_{max}	= Maximal Heart Rate
Hz	= Hertz
IET	= Incremental Exercise Test
K^+	= Potassium
Kcal	= Kilocalorie
kg	= Kilogram
$l.min^{-1}$	= Litres Per Minute
LQ	= Left Quadricep
LFES	= Low Frequency Electrical Stimulation

LH	= Left Hamstring
LG	= Left Glutael
Lga	= Left Gastrocnemius
mA	= Milliamperes
MHC	= Myosin Heavy Chain
mmHg	= Millimetres of Mercury
mmol	= Millimole
MRI	= Magnetic Resonance Imaging
mRNA	= Messenger Ribonucleic Acid
MVC	= Maximal Voluntary Contraction
N	= Newton
N_2	= Nitrogen
Na^+	= Sodium
NHS	= National Health Service
Nm	= Newton-Metre
O_2	= Oxygen
OBLA	= Onset of Blood Lactate Accumulation
Pa	= Environmental Pressure
P_B	= Barometric Pressure
PCr	= Phosphocreatine
P_{H2O}	= Vapour Pressure
Q	= Cardiac Output
RER	= Respiratory Exchange Ratio
RF	= Rectus Femoris
RQ	= Right Quadricep
RH	= Right Hamstring
RG	= Right Glutael
Rga	= Right Gastrocnemius
RPB	= Rates of Perceived Breathlessness
RPE	= Rates of Perceived Exertion
rpm	= Revolutions Per Minute
SCI	= Spinal Cord Injury
SD	= Standard Deviation
SDH	= Succinate Dehydrogenase
SEM	= Standard Error of Measurement
SM	= Semimembranosus
SR	= Sarcoplasmic Reticulum
ST	= Semitendinosus
STPD	= Standard Temperature and Pressure in Dry Air
SV	= Stroke Volume
Tb	= Expiratory Temperature
TA	= Tibialis Anterior
T_CPCO_2	= Transcutaneous Carbon Dioxide Pressure
T_CPO_2	= Transcutaneous Oxygen Pressure
TDC	= Top Dead Centre
T_r	= Room Temperature
UTI	= Urinary Tract Infection
VFT	= Variable Frequency Train
V	= Ventilation
V̇E	= Volume of Expired Air

VI	= Volume of Inspired Air
VCO_2	= Carbon Dioxide Production
VO_2	= Oxygen Uptake
VO_2 max	= Maximal Oxygen Uptake
VL	= Vastus Lateralis
VM	= Vastus Medialis
VT	= Tidal Volume
W	= Watts

Chapter 1 Introduction

It is estimated that there are 40,000 people living with spinal cord injuries in the UK. In 2001 there were 590 new patient admissions to all Spinal Injury Centres in the UK and Ireland (Spinal Injuries Association annual report, 2004). Injuries to the spinal cord include fractures, dislocations and subluxations caused by motion and trauma, with the most common trauma due to road traffic accidents and falls (Spinal Injuries Association annual report, 2004). Spinal Cord Injury (SCI) results in the complete or partial loss of sensory (afferent) and motor (efferent) function. The extent of functional alterations depends on the level of the injury and the amount of spinal cord involvement. Lesions that occur in the cervical segment (tetraplegia) cause impairment in all limbs and the trunk. Lesions at the thoracic and lumbar regions (paraplegia) retain arm function and affect the trunk depending on the lesion level. With an incomplete SCI, partial and variable sensory and motor function is retained below the level of the lesion.

An SCI will also result in the loss of autonomic nervous control below the lesion level. Spinal reflex loops however remain intact, given that a lower motor neuron injury has not occurred. As such, afferent and visceral feedback to the spinal cord is unaffected, but the transmission to the cerebral cortex is blocked at the site of injury. Similarly the sympathetic and parasympathetic outflow is blocked at the lesion level.

The implications of SCI will have vast consequences on an individual in many respects including their health-related condition. Previous research has shown that the prolonged reduction in mobility causes substantial muscular atrophy and

adaptations, reduced blood flow (Taylor et al., 1993) and a reduction in bone mineral density (Glaser, 1986) below the lesion level. Additionally, significant reductions in cardiovascular fitness (Glaser, 1986) and psychological well being (McDonald et al., 2002) have been identified. Thus SCI people are vulnerable to secondary complications such as cardiovascular disease and the development of pressure sores.

Physical activity levels in paraplegics have been identified as significantly lower than World Heath Organisation guidelines (Buchholz et al., 2003). Although upper body exercise appears to be beneficial (Durán et al., 2001) it elicits a limited rise in heart rate, due to the small amount of muscle mass activated, (Durán et al., 2001) and disuse atrophy and reduced blood flow in paralysed muscles are not prevented.

Functional electrical stimulation (FES) is used to exercise the paralysed muscles of SCI individuals. It provides the potential to reduce the long-standing detrimental effects of SCI and improve the quality of living for individuals with SCI. One case study has shown FES cycling to improve functional ability and endurance and considerably reduce medical complications, including osteoporosis, in an incomplete paraplegic (McDonald et al., 2002). It was the primary aim of this project to investigate the effects of FES cycling on physiological and health-related issues.

1.1 OUTLINE OF STUDY

Funded by the EPSRC and in collaboration with University of Glasgow and Swiss Paraplegic Centre (Nottwil), volunteers with complete paraplegia were recruited to take part in a year long study into the possible benefits of a long-term functional electrical stimulation (FES) cycling programme.

Subjects initially undertook a series of baseline tests including muscle strength and function, depth of muscle and subcutaneous fat, tissue oxygenation and seating pressures. Cardiopulmonary tests were also carried out following a period of initial muscle training that enabled the subjects to cycle. Subjects then carried out a one year cycle training programme, building up to training for one hour per day, 5 days per week. Subjects were tested at three monthly intervals during the year for cardiopulmonary fitness, muscle strength and function, depth of muscle and subcutaneous fat, tissue oxygenation and seating pressures. A PhD student in Glasgow and Post-Doctorate in Nottwil carried out all data collection on their own subjects. All data collection and analysis for the 5 London based SCI subjects and able bodied people, presented in this thesis, were carried out by the author.

1.2 EFFECTS OF SPINAL CORD INJURY

The adaptations that occur following an SCI are substantial. Spinal shock is a common occurrence during the acute phase of injury and results in temporary loss of all or most spinal reflex activity below the lesion level (for review see Atkinson & Atkinson, 1996). There is a variable effect on autonomic reflexes during this phase due to the presence of secondary neurons in ganglia outside the

spinal cord (for review see Atkinson & Atkinson, 1996). This phase ends when the spinal reflexes return, at which point the full extent of functional loss can be assessed.

The majority of subsequent physiological adaptations take place during the initial 1 to 2 years following the injury. However some adaptations, such as a reduction in bone mineral density, can take between 3 and 8 years to stabilise (Eser et al., 2004). Compared with able-bodied (AB) people, people with chronic SCI have a reduced life expectancy (McColl et al., 1997) which is primarily dependant upon their age and the level and completeness of their lesion (McColl et al., 1997; Frankel et al., 1998; Krause et al., 2004). To a lesser extent, hospitalisation time, poor health and the existence of pressure sores are determinants of an individual's life expectancy (Krause et al., 2004).

1.2.1 AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia has been identified in people with spinal lesions at the level of T6 and above i.e. above the sympathetic splanchnic outflow (T5-6). Johnson et al. (1998) reported an incidence of autonomic dysreflexia in 10 % of SCI people with injuries at or above T6 assessed over a one year period.

The condition occurs due to intact spinal reflexes responding to painful or irritating stimuli below the lesion level (e.g. bladder or bowel distension, urinary tract infections, pressure sores, spasms or external factors that stimulate pain receptors). The lack of control of sympathetic activity below the lesion level results in exaggerated sympathetic reflex responses and consequently mild to

severe hypertension can ensue. There is a subsequent fall in heart rate because of the increase in vagal efferent activity but that is not strong enough to diminish the increased blood pressure. There is also a substantial increase in sweating above the lesion level (Guttman, 1976). In people with lesions below T6, there is enough sympathetic control above the lesion to suppress this response.

Since electrical stimulation (ES) induces muscular contractions, spasms and stimulates pain receptors, it is necessary to be aware of this condition when using ES on SCI people that have never used it before. It is a potentially life-threatening condition and consequently the stimulation should be removed immediately at any sign of this occurrence.

1.2.2 MUSCULAR ADAPTATIONS

The plasticity of skeletal muscle due to modification of neural input and usage has long been established (for review see Pette, 2001). Cross-innervation experiments, as originally performed by Buller, Eccles and Eccles (1960) on cats, have established the important influence of motor nerves on the phenotypic properties of muscle (Close, 1969; Salmons & Sreter, 1976). Therefore, the complete removal of neural input activity following a complete SCI is likely to result in severe muscular adaptations.

1.2.2.1 Disuse Atrophy

A large reduction in muscle cross sectional area (CSA) has been shown to occur following SCI in both animal (Mayer et al., 1984; Lieber et al., 1986b) and human (Grimby et al., 1976; Martin et al., 1992; Greve et al., 1993; Round et al.

1993; Rochester et al., 1995b; Castro et al., 1999a; Castro et al., 1999b; Gerrits et al., 1999; Modlesky et al., 2004; Elder et al., 2004) muscle. It has been reported that a 40-45 % decline in muscle CSA occurs within 3-6 weeks of a complete SCI (Taylor et al. 1993; Castro et al., 1999a). By 6 months post injury the average CSA of affected muscle has been reported to be 45-80 % of that of age and weight matched AB people (Castro et al., 1999b). Significant increases in both subfascial and intramuscular fat have also been identified in SCI compared with AB people (Elder et al., 2004).

The extent of muscular atrophy varies in individual muscle groups, with predominantly slow muscles (e.g. soleus) reported to atrophy more than fast (e.g. gastrocnemius) in animal muscle (Mayer et al., 1984). In contrast, Castro et al. (1999a) noted a greater decline in gastrocnemius compared to soleus CSA in SCI people at 2 and 6 months post injury. The study proposed that it is not the fibre type composition of a muscle alone that determines the rate of atrophy, but possibly also its extent of use prior to the injury. Hence the majority of atrophy has been reported to occur in the larger, previously weight bearing quadriceps muscles (McComas, 1994; Gerrits et al., 1999; Taylor et al., 1993). It is likely that the rapid reduction in force production following SCI is due to the decline in muscle CSA (Rochester et al., 1995a; Gerrits et al., 1999). This has important implications for use of FES exercise, such as cycling or standing.

1.2.2.2 Fibre Type Transformation

As has been demonstrated in cross innervation studies, a slow-to-fast fibre type transformation takes place in muscles that are subjected to prolonged periods of

electrical inactivity, such as in flaccid SCI. In AB people, the motor neurones to slow muscle have continuous low frequency activity below the motor threshold (i.e. where a motor response occurs in the muscle), while those to fast motor units are silent. This low level activity over prolonged periods causes the muscle fibres to remain small, slow and fatigue resistant A complete lesion of the spinal cord results in the removal of such electrical activity below the lesion level thus a slow to fast fibre type conversion takes place.

In the initial 4-6 weeks post SCI, very little change in myosin heavy chain (MHC) isoform expression has been shown to occur (Burnham et al., 1997; Castro et al., 1999b). However, between 6 and 24 weeks post injury a conversion of type IIa to IIx fibres and an increase in fibres containing MHC IIx at the expense of IIa occurs, with no change in the relative proportion of Type I fibres or fibres containing MHC I (Castro et al., 1999b; Talmadge et al., 2002). A predominant reduction in type I fibres at 7-9 months post injury has been reported (Scelsi et al., 1982). By one year post injury, a marked predominance of type II fibres in vastus intermedius (Round et al., 1993; Greve et al., 1993), vastus laretalis (Grimby et al., 1976; Chilibeck et al., 1999), gastocnemius, soleus (Grimby et al., 1976) and tibialis anterior (Martin et al., 1992; Rochester et al., 1995b) has been demonstrated by muscle biopsy studies. Furthermore, exclusive fast MHC isoform expression in paralysed muscle has been documented at 6 years or more post injury (Andersen et al., 1996; Burnham et al., 1997). The unaffected arm muscles of SCI people have been shown to have approximately an even mixture (i.e. normal) of type I and II fibres (Grimby et al., 1976).

In AB people, the fibre type composition of a muscle has been shown to be a major determinant of the contractile properties (contraction speed and fatigability) (Harridge et al., 1996). Thus increased rates of contraction (Rochester et al., 1995a; Gerrits et al., 1999) and half relaxation (Gerrits et al., 1999) have been noted in SCI people after one year post injury.

Some variability in fibre type transformation following SCI has been identified (Hartkopp et al., 1999; Gerrits et al., 2003). In the initial 6 and 24 weeks post injury, where little or no type I to II fibre transformation or MHC expression takes place (Castro et al., 1999b), Castro et al. (2000a) identified a slowing of relaxation time in the VL muscle. It has been suggested that the changes in relaxation rate during ES might be related to sarcoplasmic reticulum (SR) calcium handling (Castro et al., 2000a; Talmadge et al., 2002) or ES induced muscle damage (Bickel et al., 2004). Some studies have also noted relaxation times in SCI people that were comparable with AB people (Martin et al., 1992; Rochester et al., 1995a), with much larger variations noted in SCI people (Rochester et al., 1995a). Rochester et al. (1995b) found an increase in the proportion of type IIx (2C) fibres in SCI people 1 to 12 years post injury suggesting the presence of ongoing fibre type transformation (Rochester et al., 1995b).

Additionally, studies have identified some individuals with chronic SCI who experience little or no fibre type transformation. Hartkopp et al. (1999) reported >99 % MHC I expression in the TA muscle of one paraplegic individual, 14

years post injury. Speed of contraction was considerably slower and fatigue resistance was remarkably improved in this individual compared with a group of SCI people with predominantly fast MHC expression. Gerrits et al. (2003) reported 3 untrained SCI people (12-29 years post injury) having a 35-62% expression of MHC I, whereas others (2.5-20 years post SCI) showed a typical 0-1 % expression of MHC I. Again, the unusually high levels of MHC I were strongly related to functional properties of the muscle, including a low fusion frequency. The reason for this variability is unclear.

1.2.2.3 Fatigue Resistance

Reduced fatigue resistance is a well-known consequence of chronic SCI and has been reported in paralysed whole muscle (Lenman et al., 1989; Rochester et al., 1995a; Gaviria & Ohanna, 1999; Gerrits et al., 1999; Castro et al., 2000a) and single motor units (Klein et al., 2006). A correlation between the percentage of type II fibres and fatigue resistance in chronically injured SCI people has been identified (Rochester et al., 1995b). This is probably due to the effect of significant adaptations that occur, concurrent with slow to fast fibre type transformation, resulting in a reduced ability of the muscle to maintain force over time.

Adaptations to the arteries are known to occur in response to altered activity of the muscles supplied (Hopman et al., 1993a; Schmidt-Trucksäss et al., 2000). The common femoral artery has been shown to exhibit significantly reduced blood flow, maximal velocity and diameter in SCI people (Hopman et al., 1996; Olive et al., 2003a), which has been reported to occur within 6 weeks after injury

(De Groot et al., 2003). Thigh blood flow in people with SCI has been noted to be as little as 65 % that of AB people (Taylor et al., 1993). Furthermore, reduced venous vascular volume, capacity and emptying rate, and increased venous flow resistance have been observed in SCI people (Hopman et al., 1994a). Such alterations are probably in response to muscle and vascular bed atrophy, inactivity of the lower limb muscle pump (Hopman et al., 1994b; 1996) and lack of sympathetic control below the lesion level (Hopman et al., 1993b; 1994b). It is therefore possible that the reduced ability of the circulation to remove accumulated lactate and hydrogen ions (H^+) would result in a rapid onset of fatigue.

Reduced capillarisation and myoglobin content are also well known adaptations with muscular disuse (for review see Pette & Vrbova, 1985). Indeed, capillary:fibre ratio has been identified to be slightly lower in SCI compared with AB people, but a large variation amongst SCI people was noted (Rochester et al. 1995b). In rat soleus muscle, Fujino et al. (2005) reported a significant reduction in capillary and anastomoses diameter following 2 weeks hindlimb unweighting. The authors noted that, based on predicted diameters required for flow of red blood cells (Henquell et al., 1976), this would result in plasma only flow in approximately 33 and 86 % of capillaries and anastomoses, respectively (compared with 1 and 30 % in control muscle). Thus the supply and capacity to uptake oxygen by a muscle would be reduced and this would also be expected to affect its fatigability. The activity of the enzyme succinate dehydrogenase (SDH), a mitochondrial enzyme, has been noted as significantly lower in SCI

people > two years post injury (Martin et al., 1992; Rochester et al., 1995b) and has been related to fatigability in SCI people (Gerrits et al., 2003).

In the initial 6 weeks post injury it has been reported that very little change in the fatigability of the paralysed soleus muscle occurs (Sheilds, 1995), despite a significant reduction in the CSA of muscle (Taylor et al. 1993; Castro et al., 1999a). This is not surprising because very little slow to fast fibre type transformation occurs during this period (Burnham et al., 1997; Castro et al., 1999b). A significantly greater reduction in fatigue resistance has been shown to occur between 6 and 24 weeks post injury (Shields, 1995; Castro et al., 2000a), but this cannot be explained by a fibre type transformation due to the persistent lack of plasticity in MHC type I in this time period (Castro et al., 1999b). It is unlikely that metabolic imbalances are the cause of greater fatigue in acute SCI because significant increases in average fibre SDH activity have been noted (Castro et al., 1999b), as well as no change in myofibrillar calcium (Ca^{2+}) adenosine triphosphate activity (Castro et al., 2000b) during this time.

Since reduced fatigue resistance has been observed in paralysed single motor units of SCI people, it is likely that impaired excitation-contraction coupling and reduced force generating ability, as opposed to ineffective transmission of electrical signals, contribute to reduced fatigue resistance (Klein et al., 2006). It has also been suggested that mechanical unloading and muscle inactivity results in higher susceptibility to contraction induced muscle damage when using ES in SCI people (Castro et al., 2000a; Castro et al., 2000b). Contraction induced muscle damage has been shown to result from unloading by hind limb

suspension in animals (Warren et al., 1994) and AB humans (Ploutz-Snyder et al., 1996). It is possible that it is this muscle damage that results in impaired excitation-contraction coupling and the reduction in fatigue resistance noted in SCI people where little or no fibre type transformation has occurred (Castro et al., 2000a; Castro et al., 2000b). Using magnetic resonance imaging (MRI), Bickel et al. (2004) found that SCI people experienced increased muscle damage compared with AB people following a single session of ES induced isometric contractions as evidenced by an elevated T_2 signal. The muscle damage remained evident 3 days after the contractions by a 22 % reduction in maximum torque (Bickel et al., 2004).

1.2.3 Cardiopulmonary Adaptations

The sedentary lifestyle following SCI leads to diminished cardiopulmonary fitness (Glaser, 1986) resulting in increased susceptibility to cardiovascular disease. Respiratory complications and heart disease have been reported as the medical diagnoses most closely associated with re-hospitalisation (Johnson et al., 1998) and the leading causes of death (Frankel et al., 1998; DeVivo et al., 1999) following SCI. In a prospective mortality study of 361 SCI males > one year post injury, Garshick et al. (2005) reported the most common primary and contributing causes of death as diseases of the circulatory and respiratory systems (40 and 24 %, respectively). Other severe secondary complications among SCI people are obesity (Janssen et al., 1996; Gupta et al., 2006), which has also been associated with increased respiratory problems (Walter et al., 2002), and diabetes (Elder et al., 2004; Garshick et al., 2005), due to an increase in body fat (Elder et al., 2004) and a sedentary lifestyle. It is likely that the

inability to activate the large muscle mass of the lower limbs and the resultant loss of functional ability is the primary cause of these complications.

1.2.3.1 Effect of SCI on physiological response to exercise

During exercise, the increased demand for oxygen by the working muscles elicits a substantial rise in oxygen uptake ($\dot{V}O_2$) and heart rate (HR), and the larger the mass of active muscle the greater the cardiopulmonary response. This response is severely reduced in SCI people because they are unable to activate the large muscle mass of the lower limbs. Furthermore, during arm exercise, SCI people have a disturbed redistribution of blood due to the lack of sympathetic innervation below the lesion level (Hopman, 1993a).

People with SCI achieve maximal oxygen uptake ($\dot{V}O_2$ max) values that are significantly lower than those achieved by AB people (Schmid et al., 1998). Approximate $\dot{V}O_2$ max values of 0.9-2.5l.min^{-1} (Coutts et al., 1983; Schneider et al., 1999; Janssen et al., 2002) and 1.5-4l.min^{-1} (Wasserman et al., 2004) have been reported for SCI people and AB people respectively. SCI people with incomplete lesions show a greater cardiopulmonary response compared with those with complete lesions (Dallmeijer & van der Woude, 2001; Janssen et al., 2002).

When comparing paraplegics and AB people exercising using their upper body only, similar $\dot{V}O_2$ max levels (2.0 and 2.3L.min^{-1} for SCI and AB respectively) have been reported (Schneider et al., 1999). Other studies have shown AB people

to attain significantly higher peak $\dot{V}O_2$ values than SCI people during maximum arm exercise (Eriksson et al., 1988; Hopman et al., 1993b; 1993c). This was probably because paraplegics are specifically trained in upper body exercise due to chronic wheelchair propulsion.

For SCI people, the disruption of sympathetic vasomotor control below the lesion level results in little or no vasoconstriction in the lower limbs to assist the redistribution of blood to the exercising musculature (Hopman et al., 1993a). This impaired response, combined with inactivity of the lower limb muscle pump results in venous pooling in the lower limbs and, consequently, reduced venous return (Hopman et al., 1992; Hopman et al., 1993a; Davis, 1993; Schmid et al., 1998a). The reduction in venous return causes the reduced stroke volume (SV) identified in SCI compared with AB people during arm exercise (Hopman et al., 1993b). It has however been shown that cardiac output (Q) is only reduced in SCI people with lesion levels above T6 (Hooker et al., 1993; Hopman et al., 1993c). SCI people with low lesion levels (≤T6) attain a similar Q to AB people by eliciting a greater rise in HR to compensate for the lowered SV (Davis et al., 1990; Hooker et al., 1993; Schmid et al., 1998a).

There is also a disturbance in thermal control below the lesion level during exercise due to the loss of autonomic nervous system control for vasomotor and sudomotor responses in paralysed parts (Sawka et al., 1989). In AB people exercising in a hot environment, increased skin blood flow to improve heat loss results in diminished venous return and thus lowered SV. This brings about a compensatory rise in HR known as 'cardiovascular drift' (Rowell et al., 1974).

This response occurs only in the sensate skin of SCI people and results in further augmentation of HR (Hopman et al., 1993c). The response is lacking below the lesion level because of the impaired cutaneous vasomotor response (Hopman et al., 1993c; Muraki et al., 1996; Yamasaki et al., 2000) that brings about the redistribution of blood to skin in response to a rise in core temperature.

The overall extent of cardiopulmonary impairment is strongly determined by an individual's lesion level and current levels of physical activity (Zwiren & Bar-Or, 1975; Davis, 1993; Muraki et al., 2000a; 2000b; Janssen et al., 2002). To a lesser extent, time since injury, age (Muraki et al., 2000a; Janssen et al., 2002) and body mass index (Janssen et al., 2002) have been identified as determinants of physical capacity in SCI people.

1.2.3.1.1 Lesion level

Lesion level has been noted as the most important determinant of physical capacity in SCI people (Muraki et al., 2000b; Janssen et al., 2002). This is probably because with ascending lesion levels, there is progressively less active muscle mass available to induce a cardiopulmonary response (Eriksson et al., 1988). Furthermore, increasing adrenergic dysfunction takes place and at key spinal levels loss of adrenal, cardiac and total sympathetic nervous control occurs (for review see Jacobs & Nash, 2004).

People with high-level lesions have been shown to have a significantly lower $\dot{V}O_2$ max than those with low-level lesions (Coutts et al., 1983; Dallmeijer & van der Woude, 2001; Janssen et al., 2002). Indeed, tetraplegics and paraplegics have

been shown to attain $\dot{V}O_2$ max values between 0.9-1l.min^{-1} and 1.5-2.5l.min-1 respectively depending on their lesion level (Coutts et al., 1983; Eriksson et al., 1988; Janssen et al., 2002). Significantly lower relative $\dot{V}O_2$ values have also been noted in high (T1-T10) compared to low (<T10) level paraplegics (Coutts et al., 1983; Muraki et al., 2000b; Janssen et al., 2002).

SCI people with lesion levels above T6 may have disturbed cardiac innervation (Hopman, 1994) as well as impaired venous return (Coutts et al., 1983; Hooker et al., 1990; Hopman et al., 1993b; 1993c; Schmid et al., 1998a) and thus reduced SV because of lower sympathetic activity than people with lesions ≤T6. Furthermore, the extent of disruption to the redistribution of blood in the lower limbs during arm exercise has been shown to be dependant upon lesion level (Hopman et al., 1993a). As such, during upper body exercise, greater reductions in leg volume have been noted in SCI people with low-level lesions (Hopman et al., 1993a). This suggests that vasoregulation of the non-exercising muscles in SCI people is induced by humoral or local reflex mechanisms and therefore is dependant upon the amount of active muscle mass. Indeed, Schmid et al. (1998a) noted higher levels of both adrenaline and noradrenaline in response to arm exercise in low-level (≤T6) SCI people compared with other SCI and AB people. The combination of these factors results in the reduced cardiac output (Q) (Davis et al., 1990; Hooker et al., 1993; Schmid et al., 1998a) and thus reduced maximal oxygen uptake observed in high compared to low-level SCI people.

High-level paraplegics (T4-T8) have been shown to elicit lower tidal volumes (VT) and higher breathing frequencies (BF) than low-level paraplegics (T11-L5).

The lower VT is probably because of the greater loss of motor control to the assistive ventilatory muscles (intercostal) of high-level paraplegics, which was compensated for by an increase in BF (Bernard et al., 2000).

The disturbance of thermal control during arm exercise noted in SCI people is also affected by lesion level, becoming further pronounced with higher lesion levels because of the increasing lack of sudomotor and vasomotor responses (Sawka et al., 1989; Hopman et al., 1992; 1993b). People with low level lesions (<T12) have shown small increases in thigh skin blood flow in response to exercise, which is not seen in those with higher lesion levels (Muraki et al., 1996). This is probably because of the enhanced cutaneous vasomotor response in SCI people with low level lesions. When exercising in heat stress conditions, increased thigh skin blood flow (Yamasaki et al., 2000) has been shown to occur in SCI people with low-level lesions only. Cardiovascular drift, which compensates for lowered SV to maintain Q in heat stress conditions, has also been shown to occur in SCI people with low-level lesions (below T6) only, thus resulting in reduced Q in people with lesions above T6, probably because of disturbed cardiac innervation (Hopman et al., 1993c).

1.2.3.1.2 Current physical activity

Although lesion level shows the strongest correlation with physical capacity, a direct and significant relationship has been identified between activity levels and physical capacity for both tetraplegics and paraplegics (Muraki et al., 2000a; Janssen et al., 2002). Janssen et al. (2002) also noted that activity level was the most important determinant of an individuals $\dot{V}O_2$ peak. However, activity levels

were assessed using a subjective scale in these studies. A more detailed evaluation of type, intensity and duration of physical activity would provide a more complete understanding of the relationship between activity levels physical capacity in SCI people.

It has been noted that, following a period of arm conditioning, small improvements in $\dot{V}O_2$ max occur in SCI people, but do not reach statistical significance (Eriksson et al., 1988; Hooker & Wells, 1989; Dallmeijer et al., 1999), possibly because of small subject numbers and heterogeneity in terms of lesion level and gender. It is also possible that exercise of such a small muscle mass is not a powerful enough stimulus to elicit central adaptations. Reduced sympathetic vasomotor tone and venous pooling would limit the exercise capacity of the upper limbs in people with SCI. Indeed, Eriksson et al. (1988) noted a significantly higher $\dot{V}O_2$ peak attained during maximal arm exercise in upper-body trained AB people compared with similarly trained SCI people (3.72 and 2.19.min^{-1}, respectively).

Peak HR during maximal arm exercise has been shown not to differ significantly between trained and untrained SCI people (Eriksson et al., 1988; Durán et al., 2001). However, Hicks et al. (2003) did show a significant increase in maximum HR following 9 months of upper body exercise training. A reduction in HR during submaximal exercise (Hooker & Wells, 1989) and during recovery from maximum arm exercise (Durán et al., 2001) has also been noted following a period of upper-body training. It is unclear whether these improvements are due

to peripheral adaptations including improved oxidative capacity of the upper body musculature or central adaptations of improved cardiac output.

Shemberger et al. (1990) noted that upper body vascular function differs significantly between paraplegics and AB people, with paraplegics showing significantly greater peak reactive hyperemic blood flow response and a larger brachial diameter at rest. Greater forearm circumferences (with similar skinfold measurements) were also noted in paraplegics. These observations suggest that adaptations occur at a peripheral as well as a central level. Significant improvements in muscle power and strength, which have been noted in SCI people following a period of upper-body training (Durán et al, 2001; Hicks et al., 2003), also suggest peripheral as well as central adaptations.

1.2.3.1.3 Time since injury

A positive correlation between the time since injury and physical capacity has been shown (Muraki et al., 2000b; Janssen et al., 2002). It is likely that the upper body adapts to the increased demands placed upon it following an SCI and thus over time improves its metabolic capacity. In line with this, improvements in physical capacity and functional ability have been noted up to 10 years post injury (Yarkony et al., 1988; Amsters et al., 2005). Tests for physical capacity using wheelchair exercise are highly specific to wheelchair users and thus the gradual improvements in efficiency over time would improve the individual's physical capacity for wheelchair exercise.

Schneider et al. (1999) noted lower respiratory exchange ratio (RER) values and a later onset of the anaerobic threshold in paraplegics compared with AB people carrying out submaximal arm cranking. This suggests that chronic daily wheelchair activity results in local adaptations of the muscles involved with upper body exercise including reduced glycogenolysis and increased rate of lipid utilisation (Schneider et al., 1999). SCI people are also likely to take up sports activities some time after an SCI (Wu & Williams, 2001), which would result in further improvements in physical capacity (Janssen et al., 2002) and metabolic adaptations of the upper body.

In people >20 years post injury, a reduced physical capacity has been identified. This was associated with a reduced ability of the upper body to perform work due to ageing, rather than time since injury (Muraki et al., 2000b).

1.2.4 Integrity of Skin Tissue

Skin tissue damage is caused by the development of local ischaemia due to pressure or tension, which eventually can result in a pressure sore. In a 5-year follow-up study, incidences of pressure sores were reported by approximately 12, 21 and 23 % of SCI people at 1, 3 and 5 years, respectively (Johnson et al., 1998). Krause (1998) noted that 46 % of 1017 SCI people reported the incidence of at least one pressure sore over a two year period. Walter et al. (2002) reported the occurrence of pressure sores in 38% of SCI people with the sacral, ischeal and trochanter areas being the primary site of occurrence. The variability between studies might be due to heterogeneity among participants. Indeed, a greater incidence of pressure sores has been reported among tetraplegics than

paraplegics and among people with complete compared to incomplete injuries (Krausse, 1998; Spinal Injuries Association annual report, 2004).

Pressure sores are a primary reason for hospital admissions (Walter et al., 2000) and hospitalisation time (Johnson et al., 1998) among SCI people thus they account for an increasing expense within the NHS (Livesley, 1990). They have also been associated with significant reductions in subjective well-being (Krausse, 1998). This is understandable because pressure sores can lead to reduced mobility and those that are not treated properly can lead to further disability (amputation) and health problems. Reduced occurrence of pressure sores has been related to lower perceptions of medical instability and higher overall fitness (Krausse, 1998). Focus should therefore be placed on both the treatment and prevention of pressure sores.

1.2.4.1 Pathophysiology of pressure sores

The viability of tissues in the skin is dependent upon an adequate supply of oxygen (O_2) and essential nutrients to the skin from the microcirculation, as well as removal of waste products by the lymphatic system.

It has been well documented that with the application of load a reduction in blood flow (Bader et al., 1986) and therefore tissue oxygenation (Knight et al., 2001) ensues, forcing tissues to draw upon their own supply of energy. When this energy supply fails, tissue damage develops. Damage to the lymphatic system due to anoxia and direct occlusion of the delicate lymphatic vessels

results in a build up of metabolic waste material within these tissues (Kosiak, 1959).

Following periods of temporary vascular occlusion, reactive hyperæmia restores the oxygen debt. Where this mechanism is absent, due to prolonged or repeated loading, cell necrosis and eventual tissue damage results (Bader et al., 1986). SCI brings about a number of factors that augments susceptibility to the development of pressure sores, subdivided into external and internal factors.

1.2.4.2 External Factors

These are any factor outside the body that contributes to the development of a pressure sore.

1.2.4.2.1 Pressure duration

The severe reduction in mobility following SCI causes specific areas of skin to become exposed to prolonged periods of unrelieved pressure. Daniel et al. (1981) reported that high pressures applied over a short duration result in muscle damage only, whereas low pressures applied for a long duration result in both skin and muscle damage. Bader & Gant (1988) identified two distinct phases during prolonged tissue loading. Phase I shows evidence of protection of the compressed soft tissue by the maintained integrity of blood flow and phase II indicates impaired protection of tissue, with relatively small increases in pressure load required to significantly reduce tissue oxygenation. This response has been well established (Newson & Rolfe, 1982; Newson et al., 1981).

1.2.4.2.2 Pressure intensity

A pressure intensity in excess of local pressure in the blood vessels is thought to cause ischaemia and a pressure of 32mmHg is widely accepted as a critical value above which local tissue damage occurs (Bennett et al., 1984). Although this value appears to be reasonable in young and healthy people (Bennett et al., 1981), there is disagreement about groups at risk of developing pressure sores, such as SCI (Bennett et al., 1984). Schubert & Farrell (1991) reported that a significantly greater load was required to occlude blood flow at the sacrum in AB compared with SCI people. Furthermore, SCI people who had no sensation required a significantly lower pressure to occlude blood flow at the sacrum than those with sensation (Schubert & Farrell, 1991). This suggests that vascular atrophy or diminished afferent and efferent nervous control results in reduced pressure tolerance around the sacrum in SCI people, particularly those with no sensation around that area. It is also possible that lower blood pressures, as noted in SCI compared with AB people, resulted in a lower pressure required to occlude the blood flow (Schubert & Farrell, 1991). Indeed, Mawson et al. (1988) noted a significant inverse correlation between systolic blood pressure and the development of pressure sores within the first month after SCI.

1.2.4.3 Internal Factors

Internal factors are those inside the body that augment the harmful effects of localised pressure.

1.2.4.3.1 Loss of sensation in paralysed parts

Normally, afferent impulses arising from the area exposed to pressure elicit signs of discomfort, numbness and pain that initiate a change of posture. In SCI these sensations are absent resulting in unusually prolonged periods of ischaemia.

1.2.4.3.2 Body composition

When pressure is evenly distributed over a large area its effects are far less than when over a small area (Garber & Krouskop, 1982; Garber et al., 1982). The considerable decline in muscle mass below the level of a spinal cord lesion (Taylor et al., 1993; Round et al., 1993), results in intensified pressure at bony weight-bearing prominences e.g. trochanter, sacrum and ischium (Kabagambe et al., 1994; Aissaoui et al., 2001).

By use of seating pressure measurements, it has been reported that SCI people have maximum pressures that are significantly greater than AB people (Thorfinn et al., 2002; Gutierrez et al., 2004). It has also been reported that SCI people have a significantly smaller overall contact area than AB people, despite being similar in weight (Gutierrez et al., 2004). In contrast, Thorfinn et al. (2002) noted no significant difference in overall contact area between SCI and AB people, but the ranges for SCI people were large in this study and the SCI group were somewhat older than the controls (mean age 28 and 55 years for AB and SCI people respectively) which might influence their body size, however height and weight were not reported. When measured on a hard surface, SCI people distribute load more asymmetrically than AB people, probably due to the loss of sensory ability and perception (Gutierrez et al., 2004). Loading was noted as more symmetrical when measured in the subject's own wheelchair (Gutierrez et al., 2004).

1.2.4.3.3 Reduced blood flow and tissue oxygenation

Significant reductions in the common femoral artery blood flow and diameter have been reported to occur after chronic SCI (Taylor et al., 1993; Hopman et al., 1994; 1996; 2002; De Groot et al., 2003; Olive et al., 2003a). Following chronic denervation or muscle disuse, reductions in the number of capillaries (Tyml et al., 1999; Dedkov et al., 2002; Wagastuma et al., 2005) and capillary:fibre ratio have also been reported in animal (Desplanches et al., 1990; 1991; Tyml et al., 1999; Kano et al., 2000; Dedkov et al., 2002; Fujino et al., 2005; Wagastuma et al., 2005) and human (Carpenter & Karpati, 1982) muscle. However, there is an increase in capillary density because the extent of muscle fibre atrophy is greater than that of capillary degeneration following muscular disuse or denervation (Carpenter & Karpati, 1982; Desplanches et al., 1990; 1991; Hudlicka et al., 1992; Fujino et al., 2005; Kano et al., 2000). There is also evidence of capillary necrosis or damage in disused animal (Tyml et al., 1995) and denervated human muscle (Carpenter & Karpati, 1982). A decrease in luminal diameter of both the capillaries and interconnecting anastomoses has also been reported following hindlimb unloading in rat soleus muscle (Kano et al., 2000; Fujino et al., 2005). These consequences of muscular disuse are likely to augment the harmful affects of any given pressure load (Guttmann, 1976; Rodriguez & Claus-Walker, 1988).

During acute SCI, there is a reduction in vascular resistance and vasodilation as well as a reduction in blood pressure (Triolio & Bogie, 1999). This might impair blood circulation, lymphatics (Krouskop et al., 1978; Krouskop, 1983) and tissue resistance under load (Guttmann, 1976) during the acute stages of SCI. Indeed,

the occurrence of pressure sores has been shown to be inversely related to systolic blood pressure during the initial 30 days after SCI (Mawson et al., 1988). It is less clear whether this effect exists in long term SCI. It has been shown that vascular resistance is significantly higher in chronically injured SCI people compared with AB people (Hopman et al., 2002; Koojman et al., 2003). Hopman et al. (2002) found that arterial blood flow was significantly lower in the SCI group whereas mean arterial blood pressure was similar in both groups resulting in increased vascular resistance in the SCI group. It is possible that increased α-adrenergic tone contributes to the increased vascular resistance observed in chronic SCI (Koojman et al., 2003).

Patterson et al. (1993) reported that SCI people had significantly reduced blood flow, measured by Laser Doppler, under no load, with the application of 30 and 75 mmHg loads and during recovery from loading. The reactive hyperaemic response, a consequence of temporarily occluded blood flow, is a good measure of the ability of an individual to recover from tissue hypoxia. Olive et al. (2003a) reported that blood flow recovery after ischemia was significantly prolonged in SCI compared with AB people. Vascular reactivity is particularly important for SCI people during pressure relief (wheelchair pressure lifts). It has been reported that following a period of sitting hyperaemia is more intense over the ischial tuberosities for both AB and SCI people, however absolute values were higher for AB people (Thorfinn et al., 2002). There was no correlation between the relative hyperaemia intensity and pressure created during sitting, although the authors did identify a critical pressure above which the hyperaemic response was greater (Thorfinn et al., 2002). Schubert & Farrell (1991) noted a significantly

lower peak skin blood flow over the gluteus muscle following occlusion in SCI compared with AB people. They also noted that time to peak blood flow was significantly prolonged in SCI people (Schubert & Farrell, 1991).

Studies to investigate whether paralysed parts have lowered oxygenation have mostly investigated oxygen tension in the superficial layers of the skin. Bogie et al. (1992) measured transcutaneous oxygen and carbon dioxide pressures (T_CPO_2 and T_CPCO_2, respectively) at the sacrum of acute SCI people in a supine position. The study showed that CO_2 levels increased as O_2 decreased under load, with a subsequent recovery in O_2 levels. The recovery in O_2 levels suggests that the mechanism whereby CO_2 stimulates the nervous system to dilate arterioles and drive blood flow to anoxic areas remains intact during acute SCI (Bogie et al., 1992). Similar recovery of O_2 in response to the removal of load has been noted following chronic SCI (Kabagambe et al., 1994).

1.2.4.3.4 Tissue Oxygenation

It has been reported that T_CPO_2 values under no load are significantly reduced in SCI compared with AB individuals (Mawson et al., 1993; Liu et al., 1999). Conversely other studies have demonstrated no significant differences in T_CPO_2 values between SCI and AB people under similar conditions (Patterson et al., 1993; Kabagambe et al., 1994; Peters, 2000). These discrepancies could be attributed to variability between and/or within subjects due to factors such lesion level (including whether paralysis is spastic or flaccid), time since injury, body size and configuration (muscle mass:adipose tissue ratio) and training status, which might affect an individuals T_CPO_2. Indeed, Peters (2000) noted that flaccid

SCI tended to result in lower T_CPO_2 levels. Alternatively, natural day-to-day variation or methodological issues might explain such inconsistent results.

Patterson et al. (1993) noted that SCI people experience a significantly greater reduction in T_CPO_2 under a load of 30 mmHg than AB people. This indicates impaired vasomotor control under lower loads for SCI people, although the authors did note that some SCI people showed responses comparable to AB, suggesting a variable response among this population. A possible explanation is that people with lesion levels <T6 have been shown to exhibit lower T_CPO_2 values compared with those with higher lesions in response to similar loads, for longer periods of time during the acute phase after SCI (Bogie et al., 1995). The study suggested that people with lesions <T6 are more likely to have suffered lower motor neuron damage resulting in loss of reflex activity (flaccid paraplegia), which causes greater reductions in muscular tone and atrophy below the lesion level compared to other SCI people. Thus reduced soft tissue surrounding the capillary bed reduces the amount of pressure the capillaries can withstand, resulting in lower T_CPO_2 under load in people with flaccid paraplegia (Bogie et al., 1995). Indeed, Peters (2000) reported that people with flaccid SCI tended to require a smaller load to reduce T_CPO_2 to 10 mmHg compared with other SCI and elderly AB people. Under greater load (75 mmHg), no significant differences in T_CPO_2 values have been observed between SCI and AB people (Patterson et al., 1993). The amount of load required to reduce T_CPO_2 to a critical threshold (10 mmHg) has also been reported to be similar between SCI and AB people (Kabagambe et al., 1994).

It has been noted that SCI people have a slower rate of recovery to pre-load T_CPO_2 values following the removal of load, and that some are unable to recover to pre-load values after 5 minutes of pressure relief (Kabagambe et al., 1994). Peters (2000) however reported no correlation between subject group and time to 50 % recovery of preload T_CPO_2 tension in spastic and flaccid SCI and healthy elderly people.

1.3 SUMMARY

Spinal cord injury, a relatively common occurrence in the UK, severely impacts on many aspects of life including an individual's health related status. Loss of sensation and motor control below the lesion level results in reductions in muscle bulk and blood flow below the lesion level, increasing an individual's susceptibility to pressure sores. Reduced cardiopulmonary fitness, due to the inability to exercise the large muscle mass of the lower limbs, leads to a greater susceptibility to cardiovascular disease, obesity and diabetes. Decreased bone mineral density also increases the risk of fractures.

Although upper body exercise is easily available for SCI people it fails to induce significant cardiopulmonary adaptations, does not involve the parts of the body affected by the lesion and increases the possibility of shoulder joint damage. Thus functional electrical stimulation (FES) exercise holds the possibility of increasing muscle bulk and blood flow in the lower limbs and to increase cardiopulmonary responses to exercise by inducing exercise below the lesion level.

1.4 AIMS OF STUDY

The primary aims of this study were to identify the effects of an intense, long-term programme of FES cycling for SCI people on

- muscular size, strength and function
- power output, endurance and cardiopulmonary fitness during FES cycling
- susceptibility to pressure sores

The study also aimed to identify limitations in current FES cycling systems and to propose how they might be improved to allow FES cycling to become more appealing, realistic and an effective option for SCI people.

The following chapter provides a summary of current literature in FES cycling, describes how FES cycling works and its potential benefits and outlines the training programme that was carried out for this study. Results presented include time spent cycling and compliance to the set training programme.

Chapter 2 Functional Electrical Stimulation for Cycling: Training Programme and Outcome Measurements.

This chapter provides an overview of the training study. Previous literature in functional electrical stimulation (FES) cycling training studies is reviewed, including the potential health benefits for people with SCI. FES cycling and the training programme carried out for this study are described. Results presented and discussed include training compliance and the amount of training carried out by each subject.

2.1 Literature Review

Petrofsky and co-workers (1982; 1983; 1984) first described the development of a computerised, closed loop control stimulator and cycle ergometer for people with spinal cord injury (SCI). Electrical stimulation (ES) is used to induce muscular contractions in paralysed muscle groups allowing them to work against a load in order to build muscular strength (Petrofsky, 1987). This also has the potential to allow people with SCI to exercise at a level that stresses their cardiopulmonary system. Petrofsky (1987) reported that the incidence of pressure sores, fractures, kidney and bladder infections and thrombophlebitis over a one year period, were reduced in people with SCI who carried out 15 minutes of ES cycling three times per week. This system is referred to as functional electrical stimulation (FES) cycling.

Much work has since been carried out investigating the potential benefits of FES cycling for people with SCI. A number of studies have carried out short-term (3-

6 months) programmes of FES cycling, training 2-3 times per week (Pollack et al., 1986; Ragnarrson et al., 1988; Pollack et al., 1989; Arnold et al., 1992; Faghri et al., 1992; Goss et al., 1992; Hooker et al., 1992; Petrofsky & Stacy, 1992; Sköld et al., 2002). These studies have used electrical stimulation of the quadriceps, hamstrings and gluteal muscles in a synchronised pattern to cycle a static ergometer.

2.1.1 POWER OUTPUT DURING FES CYCLING

It has been consistently reported that people with complete spinal lesions gain improvements in both muscle power (post-training peak power output 0-55 Watts (W)) and endurance (increasing pedalling duration up to 30 minutes) following an FES cycling programme. Improvements in power output and endurance as well as considerable improvements in physical function have also been reported in people with an incomplete SCI (Sloan et al., 1994; Donaldson et al., 2000). However, the power output produced during FES cycling, remained substantially lower than that of untrained able-bodied (AB) people (approximately 250-400 W) and lower than the power outputs required to cycle outdoors.

Goss et al. (1992) reported that 5 SCI people achieved 6-12 W following 6 months training and Faghri et al. (1992) reported that 6 paraplegics were able to maintain an average of 17 W over 30 minutes following three months training. Hooker et al. (1992) reported training power outputs of between 0 and 31 W in 18 SCI people after three months training. In the same study, peak power output in a graded exercise test increased from 13.6-19.7 W. Mutton et al. (1997) reported that the average power output attained in a graded FES cycling exercise

test was 14 W (following 18 weeks of FES cycling, training two times per week) which increased significantly to 18 W following a further 25 weeks of hybrid (arm + FES leg cycling) exercise.

In a long-term FES training study (one year), Mohr et al. (1997) reported considerable variation in initial performance with power outputs of 0-18 W after 6 months of training with no further improvements in the final 6 months. The study also reported peak power outputs attained during training of 6-42 W and these were attainable for no longer than 7 minutes.

The few studies that have investigated the effects of a more intense training programme of FES cycling in people with SCI have reported a similar range of power outputs. Pacy et al. (1987) achieved power outputs of 6-19 W in 4 paraplegics who carried out FES cycling 5 times per week for just 15 minutes. Hjeltnes et al. (1997) trained 5 SCI people 7 times weekly for 8 weeks. Peak power output achieved during a graded FES cycling exercise test after training was 22 W. The effects of a more intense training programme over a longer period have not yet been investigated.

Theisen et al. (2002) measured power output during 40 minutes of FES cycling in 5 SCI people and noted a distinct pattern. Peak power output was achieved after 2 minutes (10.7 W), this then declined and subsequently recovered to a level slightly lower than the peak power (5.3, 8.2 and 6.1 W at 6, 20 and 40 minutes respectively).

Previous studies have highlighted considerable inter-individual variation in the power outputs achieved (Ragnarrson et al., 1988; Mohr et al., 1997; Gerrits et al., 2000) as well as a particular difficulty in increasing cycling duration and intensity in some SCI people (Arnold et al., 1992). The reasons for this remain unclear. It is possible that sex, age, level of lesion, time since injury (Arnold et al., 1992; Gerrits et al., 2000), degree of muscular atrophy (Mohr et al., 1997) and type and intensity of activities carried out pre-injury all contribute to the variations identified. Furthermore, muscular nerve distribution and the possibility of peripheral nerve damage are likely to differ between individuals (Kagaya et al., 1996). Stimulation parameters, electrode size, position and impedance might also cause individual variation in the response to FES cycling.

Peak oxygen uptake (VO_2) values determined during FES cycling have been noted to exceed the predicted values based on power output (Goss et al., 1992). For example, it is predicted that AB people cycling voluntarily at power outputs of 50-400 W, attain peak VO_2 values of 0.9-5.7 $L.min^{-1}$ (Åstrand et al., 2003). SCI people however have been reported to achieve peak VO_2 values from 0.9-2.5$l.min^{-1}$ (Coutts et al., 1983; Schneider et al., 1999; Janssen et al., 2002) at power outputs of 10-25 W. It is important to identify whether these relatively low power outputs and high VO_2 values are a consequence of physiological adaptations that occur due to the SCI or due to inefficiencies in the application of electrical stimulation. It is likely that the known consequences of chronic SCI (eg. reduced muscle size and strength, reduced blood flow and oxidative capacity of muscle resulting in diminished fatigue resistance) would result in a reduced ability to develop and maintain muscle force. Reversal of some of these

adaptations has been shown to occur in response to a period of low frequency ES training in SCI people (Gerrits et al., 2002; 2003). Fatigue resistance and speed of contraction have been shown to return to levels comparable to AB people (Gerrits et al., 2000; 2002). Muscle strength and power output however remained significantly lower for SCI people using ES than that achieved by voluntary activation in AB people. When AB people performed FES cycling following epidural anaesthesia power outputs of 20-40 W were achieved compared with 70-120 W during voluntary cycling at a similar VO_2 and heart rate (Kjær et al., 1994). This indicates that the use of ES to induce exercise is considerably less efficient than voluntary exercise in AB people.

FES cycling in SCI people elicits higher metabolic rates than in AB people exercising with voluntary contractions at similar absolute work rates (Barstow et al., 1995). A number of observations are indicative of a greater contribution of anaerobic pathways to energy production during ES exercise. Significantly greater increases in the concentrations of blood and muscle lactate, plasma potassium and hydrogen ions have been noted from electrically stimulated compared with voluntary exercise in AB people (Kjær et al., 1994; Kim et al., 1995a; Hamada et al., 2004). Similarly, higher lactate levels have been found during graded FES leg cycling compared with graded arm ergometry in people with SCI (Hjeltnes et al., 1997). Mohr et al. (1997) reported blood lactate values of 10.3 mmol in SCI people after a graded FES cycling test. High respiratory exchange ratio (RER) values (0.9-1.3) have been reported for SCI people carrying out FES cycling (Hjeltnes et al., 1997; Mohr et al., 1997).

Consequently metabolic efficiency is considerably lower when carrying out electrically stimulated than during voluntary exercise. Glaser et al. (1989) compared the efficiency of FES cycling in SCI people with voluntary cycling by AB people at power outputs of 6-42 W. Efficiency ranges were 2-14 % and 4-34 % for SCI and AB people respectively. Other studies have reported metabolic efficiency during FES cycling in SCI people of 2-5 % (Goss et al., 1992; Petrofsky & Stacy, 1992). Metabolic efficiency during voluntary cycling is commonly reported to be 20-29 % in AB people (Åstrand et al., 2003).

There are a number of reasons to explain the considerably reduced efficiency during FES cycling. Firstly, it has long been suggested that the natural recruitment order of muscle fibres during voluntary activity is reversed with electrical stimulation, with fast motor neurons possibly being recruited first because of their large motor neurons. This effect would be further exaggerated by a conversion towards the more fatigable type II fibres following SCI (Grimby et al., 1976; Martin et al., 1992; Round et al., 1993; Greve et al., 1993; Rochester et al., 1995b; Chilibeck et al., 1999). Further support for this comes from the greater levels of fatigue that have been identified during ES in SCI than AB people (Gerrits et al., 2001a; Olive et al., 2003b). However, it has recently been suggested that this principle may not apply to humans (Gregory & Bickel, 2005).

The lower level of fatigue seen during voluntary exercise might be due to asynchronous firing of motor units and the recruitment of additional motor units in order to maintain force. Adams et al. (1993) reported that ES stimulates the same fibres repeatedly (determined by the magnetic resonance signal T_2)

resulting in increased metabolic demand (Adams et al., 1993). In addition, firing rates are reduced to compensate for fatigue induced mechanical slowing during voluntary exercise (Carpentier et al., 2001). The fixed pattern of ES does not allow any compensation for fatigue in terms of muscle recruitment or firing frequency, and thus fatigue is likely to be compounded. For FES cycling, it is worth noting that the majority of studies activate just 3 or 4 muscle groups (usually quadriceps, hamstrings, gluteals and gastrocnemius) in a synchronised pattern to elicit a cycling motion. However a number of additional muscles are used in voluntary cycling as stabilisers or synergists. The sequencing patterns of the quadriceps and hamstring generally consider these muscles as monoarticular, functioning as knee extensors and flexors, respectively, whereas these muscle groups actually have different actions on the hip and knee. Thus the stimulation timing of these muscle groups might result in them working to some extent as antagonists, providing a negative contribution to power output.

2.1.2 POSSIBLE HEALTH BENEFITS OF FES CYCLING

Improvements in cardiopulmonary fitness have been reported following both short-term (Pollack et al., 1986; Pollack et al., 1989; Arnold et al., 1992; Goss et al., 1992; Hooker et al., 1992; Hjeltnes et al., 1997; Kjær et al., 2001a) and long-term (Mohr et al., 1997; Mutton et al., 1997) FES cycling programmes. These have been reported to plateau after 6 months of training (Mohr et al., 1997). However some studies have shown no changes in cardiopulmonary fitness following a period of FES cycling (Ragnarrson et al., 1988, Faghri et al., 1992; Petrofsky & Stacy, 1992) and the reasons for the discrepancy between studies

remain unclear. It is possible that differences in the training programmes implemented and in the methodology used for testing VO_2 contributes to this.

Short-term training has been shown to bring about significant increases in peak heart rate (Pollack et al., 1989, Faghri et al., 1992; Hooker et al., 1992) and cardiac output (Faghri et al., 1992; Hooker et al., 1992) as well as reductions in submaximal heart rate (Faghri et al., 1992; Mutton et al. 1997). It has been suggested that improved cardiac volume loading is brought about by improved venous return due to the activation of the venous muscle pump with FES cycling (Faghri et al., 1992).

It has consistently been reported that significant increases in muscle size (Petrofsky, 1987; Pacy et al., 1988; Arnold et al., 1992; Mohr et al., 1997; Hjeltnes et al., 1997; Scremin et al., 1999; Sköld et al., 2002), fibre cross sectional area (Crameri et al., 2002) or diameter (Neumayer et al., 1997) and strength (Ragnarsson et al., 1988; Hjeltnes et al., 1997; Mohr et al., 1997; Gerrits et al., 2000) occur following both short and long-term programmes of FES cycling. Pacy et al. (1987) noted that the significant increases in bilateral quadriceps muscle area appeared unrelated to the power outputs attained by the paraplegics during FES cycling. The reason for this discrepancy is unclear.

Increased muscle bulk may bring about improved perceptions of self-image for people with severely atrophied muscles following an SCI (Sipski et al., 1989). Furthermore, improved muscle bulk in the guteal muscles might be effective in

reducing seating pressure around bony areas such as the ischeal tuberosities, reducing the susceptibility to pressure sores.

Hjeltnes et al. (1997) showed a significant reduction in body fat following an intense FES training programme lasting 8 weeks. However, other studies have shown no changes in adipose tissue (Scremin et al., 1999; Sköld et al., 2002).

Improved fatigue resistance in paralysed muscle has also been reported following ES training (Stein et al., 1992; Gerrits et al., 2000; Rochester et al., 1995a; Gerrits et al., 2002) due possibly to fast-slow fibre type transformation (Greve et al., 1993; Anderson et al., 1996; Crameri et al., 2002) and increased capillary density (Salmons & Henriksson, 1981). Indeed, a period of FES cycling has also been shown to reverse the vascular atrophy and reduced lower limb blood flow that occurs due to reduced activity in paralysed limbs (Gerrits et al., 2001a; Crameri et al., 2002). These adaptations are likely to contribute to the improved cycling performance noted following a period of FES cycling by enhancing oxygen delivery and removal of metabolic products. It is also possible that these adaptations will improve the microcirculation in paralysed parts leading to improved tissue health and a reduced susceptibility to pressure sores.

These potential health benefits will be discussed in more detail in subsequent chapters of this thesis.

2.1.3 PERCEPTIONS AMONG THE SCI POPULATION

In 1989 Sipski et al. conducted a survey to investigate the perceptions of SCI people who had carried out FES cycling as part of a rehabilitation programme. Sixty two and 54 % of subjects reported improved perceptions of self-image and appearance respectively. Other perceived changes included a decrease in lower limb oedema and improved bladder and bowel functioning (Sipski et al., 1989). Another study noted that subjects reported increased feelings of well being, higher energy levels and reduced fatigue after an FES cycling programme (Ragnarrson et al., 1988). Sipski et al. (1993) investigated the long term use of FES cycling in people with SCI when they had a system freely available to them in their own home. The study identified 19 out of 28 people as regular FES users. They also noted adherence to FES cycling was significantly related to the sex of an individual and pre-injury exercise habits, with men and pre-injury exercisers more likely to continue with FES cycling.

2.1.4 CONTRAINDICATIONS OF FES CYCLING

No adverse effects of FES cycling (skin reactions or bone fractures) have been reported by any of the studies mentioned above although Hartkopp et al. (1998) reported a bone fracture in one person with SCI during isometric ES of the quadriceps muscle. It was suspected that increased muscular strength combined with severe osteoporosis contributed to the fracture. Sipski et al. (1989) reported that 66 % of SCI people that experience neurogenic pain noted an increase in this pain when carrying out a programme of FES cycling.

2.2 FES CYCLING – HOW IT WORKS

FES cycling, described in detail by Perkins et al. (2001), involves stimulation of lower limb muscles to elicit a cycling motion. The FES system (Fig. 2.1) consists of a commercially available recumbent tricycle (ICE Crystal Engineering, USA) adapted for paraplegic users. Recumbent trikes were chosen because they provide stability, reduce peak seating pressures (by distributing body weight over a relatively large surface area) and are more easily accessible for people with SCI as the handlebars are removable for ease of transfer. Orthoses are attached to the pedals to hold feet on the pedals and help prevent abduction and adduction of the legs during cycling.

A 10-bit shaft encoder is attached to a cogwheel and driven by a chain connected to the left crank (Fig. 2.2). The encoder takes measurements of the crank angle and activates the stimulator via a junction box. By means of computer software, the stimulator is programmed to stimulate the required muscles at a specified time and intensity to produce a cycling motion. The encoder angles for each muscle group were set using a protocol by Perkins et al. (2001). Three subjects were tested for torque produced by each muscle group at a number of static pedal angles. Where the torque produced by any given muscle was > 1 Nm, the muscle group was determined as 'on' for that pedal angle (Perkins et al., 2001). The encoder angles are set slightly in advance of the specified 'on' angle for each muscle group to compensate for a time delay that occurs between the crank angle being reached and the required muscle receiving the stimulation and eliciting a muscular contraction. A throttle has been fitted to the trike for control of

stimulation intensity. The tricycle can be used outdoors or mounted on an exercise trainer for indoor use.

FIG. 2.1: RECUMBENT TRICYCLE USED BY SCI SUBJECTS FOR FES CYCLING WITH STIMULATOR ATTACHED.

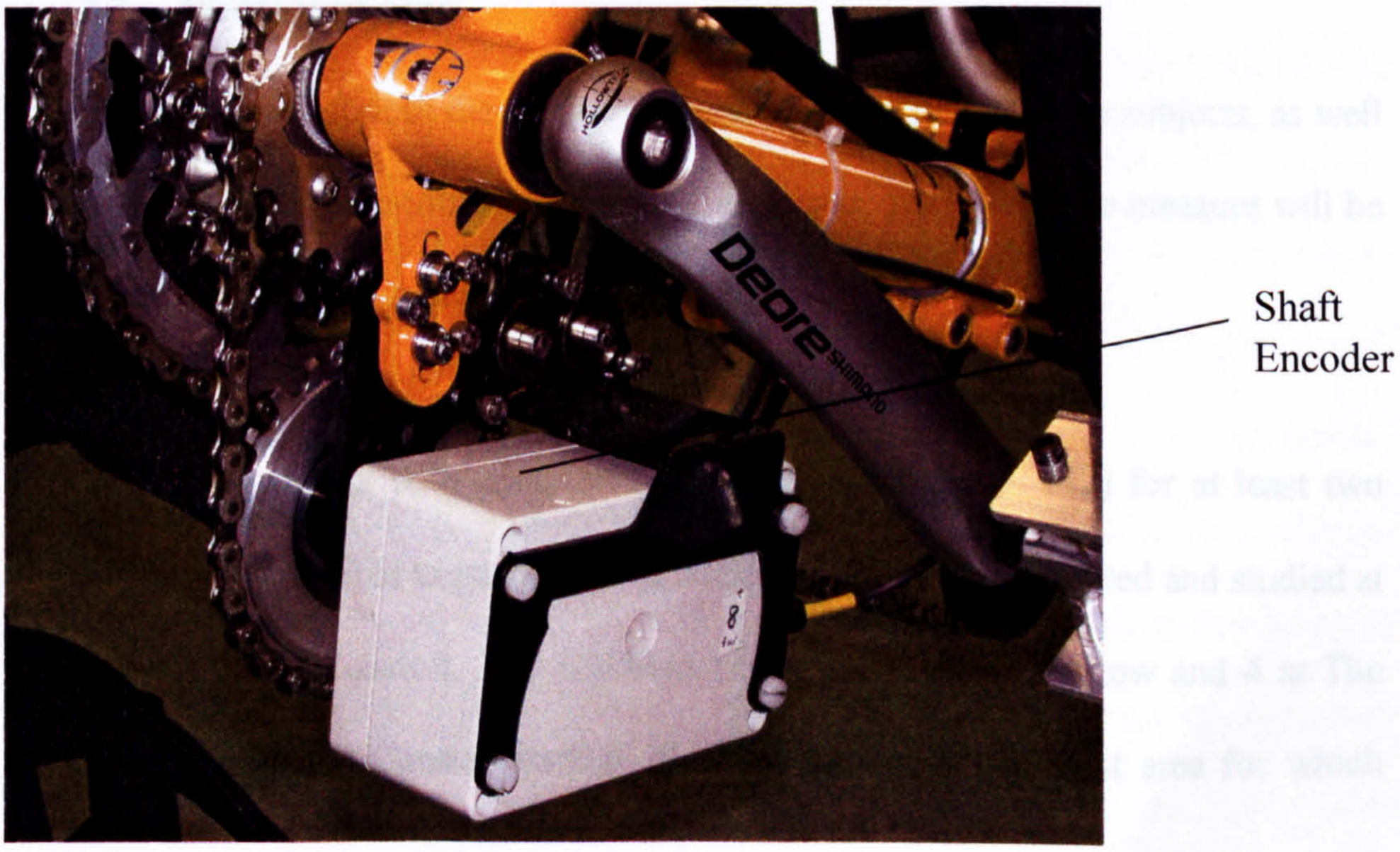

FIG. 2.2: SHAFT ENCODER ATTACHED TO LEFT FRONT CRANK OF TRICYCLE.

Adhesive electrodes (PALS Platinum, Axelgaard, USA) were used on 4 muscle groups. Oval electrodes (12.5 x 7.5 cm) were positioned proximally and distally on the lateral and medial aspects of the quadriceps, respectively, and proximally and distally on the midline of the hamstrings. Round electrodes (7 cm) were positioned proximally and distally on the midline of the gluteus medius and proximally on the gastrocnemius muscles. Small round electrodes (5 cm) were used for the distal gastrocnemius. Electodes sizes were altered depending on muscle size. Stimulation on and off angles are given in Table 2.1, where left hip most flexed represents 0°.

Angle	LQ	LH	LG	LGa	RQ	RH	RG	RGa
On (degrees)	6	113	31	141	186	294	212	322
Off (degrees)	102	212	124	226	282	31	305	45

TABLE 2.1: STIMULATION ON AND OFF ANGLES FOR RIGHT (R) AND LEFT (L) QUADRICEPS (Q), HAMSTRINGS (H), GLUTEALS (G) AND GASTROCNEMIUS (GA) USED DURING FES CYCLING WHERE LEFT HIP MOST FLEXED REPRESENTS 0°.

2.3 METHODOLOGY

This section describes the training programme completed by the subjects, as well as an outline of the timing of outcome measures. Each outcome measure will be described in detail in subsequent chapters.

Eleven volunteers with complete paraplegia (level T3 to T12) for at least two years were recruited to participate in the study. Five were recruited and studied at Kings College London, 2 at Southern General Hospital, Glasgow and 4 at The Swiss Paraplegic Centre, Nottwil. Each centre had a specialist area for which

they collected and analysed data from all 11 subjects. This thesis presents data from the 5 London subjects only.

Inclusion and exclusion criteria are outlined in Table 2.2. Subjects initially underwent a series of baseline tests including bone mineral density, muscle strength and spasticity, thickness of muscle and subcutaneous fat, tissue oxygenation and seating pressures.

INCLUSION CRITERIA	EXCLUSION CRITERIA
- Aged 18-65 years. - Lesion level ≤T3 for >2 years. - No additional medical or psychiatric complications. - Sufficient range of motion at the joints. - .Bone trabecular densities above 40 mg/cm^3 in the distal tibia and femur. - Willingness to exercise according to the exercise programme.	- Any previous or current involvement in FES exercise. - Failure to transfer safely between wheelchair and trike. - Failure to build muscle strength required for carrying out cycling following a period of initial training. - Excessive spasticity

TABLE 2.2: INCLUSION AND EXCLUSION CRITERIA FOR FES CYCLING STUDY.

2.3.1 INITIAL TRAINING

Initial training took place until subjects were strong enough to cycle with no load applied to the back wheel. Each subject was set up with a Stanmore Stimulator (Phillips et al., 1993) to carry out initial muscle training at home on a chair or the edge of a bed. The training involved simultaneous stimulation of the quadriceps, glutei and gastrocnemius to produce knee and hip extension and of the contra-lateral hamstrings to produce knee flexion. This produced alternate hip and knee extension and flexion with each contraction lasting 6 seconds followed by 2 seconds rest. Each subject was taught correct placement and care of electrodes. Stimulation current and pulsewidth range were set at a level that produced a

maximal contraction determined by palpation of the muscle group at a stimulation frequency of 20 Hz. Subjects were asked to stop each training session when their muscles fatigued (defined as no visible contraction of the quadriceps muscle group) and to gradually build training to five 30 minute sessions per week over a period of six weeks. Once 30 minutes training was achieved, subjects were asked to add 1 kg ankle weights incrementally. After 6 weeks training subjects completed a cycling trial. Once the following criteria were met each subject progressed to cycle training:

- Able to cycle for 10 minutes (unassisted) at a resistance of 5 W.
- Able to transfer from a wheelchair to the tricycle with reasonable safety.
- Able to place the electrodes correctly and understand how to use the stimulator, including how to connect the electrodes correctly.

Prior to the start of cycle training, baseline cardiopulmonary tests (details in Chapter 4) were completed. Subjects were then given a trike (Trice, Inspired Cycle Engineering, UK), ergotrainer (Tacx Flow ergotrainer, Wassernaar, Netherlands), stimulator (Stanmore, UK), surface electrodes (PALS Platinum, Nidd Valley Medical Ltd, UK) and cables to set up in their own home.

2.3.2 Cycle Training

The cycle training programme lasted a total of 52 weeks for each subject. Subjects were asked to maintain a cadence of 45-55 revolutions per minute (rpm) during all cycle training. Stimulation current and pulse width for each muscle group was set as described above. All cycle training was carried out at 50 Hz unless this caused rapid fatigue, in which case cycle training began at 20 Hz.

Subjects were asked to record date, time, duration, power and cadence of each session carried out (Appendix 4). Subjects were provided with support and advice by regular (once weekly) telephone conversations. Home visits were also carried out (as required) to monitor training and resolve equipment problems. Subjects were allowed to carry out training sessions outdoors if they chose to.

2.3.2.1 Training Frequency

The number of training sessions per week was set as follows: three sessions per week during weeks 1–8, 4 sessions per week during weeks 9–16 and 5 sessions per week during weeks 17–52. In total, 232 sessions were scheduled for the one year programme. When > one week of training had been missed due to holiday or illness, additional weeks were added.

2.3.2.2 Duration

Subjects were asked to carry out the initial three sessions with no load on the back wheel, and record the duration achieved before fatigue (defined as cadence falling below 45rpm). The highest duration achieved during this period was set as the initial cycling duration. Subjects were then asked to increase their target duration for the session by 10 minutes every three weeks until 60 minutes cycling was attained.

2.3.2.3 Resistance

Resistance was adjusted using the ergotrainer and 3 x 9 gears. The back wheel of the trike was mounted on the ergotrainer, which was attached to the back wheel

to apply magnetic brake resistance. There are 14 settings ranging from -4 (lowest) to +9 (highest). The trainer setting and cycling cadence calculated power output. At 50 rpm, there is approximately a 1-2 W increment between each trainer setting. Subjects were asked to begin training with three sessions at no load. Following this, they were asked to attempt the lowest load (trainer setting -4) and continue until fatigue (defined as cadence ≤ 45 rpm), then continue cycling with no load until fatigue or until the target duration for the session was completed. Once 10 minutes continuous cycling at -4 was achieved for at least three consecutive sessions, then attempted the next resistance (-3) until fatigue at which point they resumed cycling at -4. Progression through the resistance settings (-4 to +9) was continued in this way throughout the 52 week training programme. Once +9 had been achieved, gears on the trike were used to continue increasing the resistance.

2.4 Outcome Measures

During the 52 week cycle training programme, subjects were tested at 13, 26, 39 and 52 weeks. At each time-point the following measurements (described in detail in subsequent chapters) were made:

- Cardiopulmonary fitness: (i) Incremental exercise test (IET) and (ii) Constant load exercise test (at 70 % maximal power achieved during the baseline IET).
- Contractile properties of the quadriceps: (i) Maximal isometric strength, (ii) Speed of twitch, (iii) force:frequency relationship and (iv) Fatiguability.
- Muscle and fat thickness of the quadriceps, hamstrings, gastrocnemius and glutei: (i) Magnetic Resonance Imaging (MRI) (at baseline and 52 weeks only) and (ii) Ultrasound.

- Tissue oxygenation: (i) Transcutaneous oxygen tension (T_CPO_2) under load at the sacrum.
- Seating pressures: Maximum pressures under each ischeum in (i) a standard NHS wheelchair and cushion (ii) the subjects' own wheelchair and cushion.

2.5 RESULTS

Ethical consent for this study was obtained from King's College Hospital Local Ethics Committee and King's College London.

2.5.1 SUBJECT DETAILS

Five subjects (1 female) gave informed consent to participate in the study. Average (± SEM) age and approximate height and weight (predictions made by subjects due to unavailability of wheelchair accessible scales) at the start of the study were 45.2 ± 3.4 years, 173.04 ± 9.37 cm and 71.88 ± 12.05 kg, respectively. Individual characteristics at the start of the study are given in Table 2.3.

Subject	Sex	Age (years)	Height (cm)	Weight (kg)	BMI	Lesion level	Time since injury (months)
1	Female	45	162.5	53.5	20.1	T9	52
2	Male	58	173.0	84.0	28.1	T4	116
3	Male	39	169.0	79.0	27.7	T3	111
4	Male	40	188.0	66.7	18.9	T4/5	130
5	Male	44	172.7	76.2	25.5	T4	48

TABLE 2.3: DETAILS OF THE FIVE LONDON SUBJECTS WITH COMPLETE SCI AT THE START OF THE STUDY (BMI = BODY MASS INDEX).

2.5.2 COMPLIANCE

All 5 subjects completed the training programme. The average time taken was 60.6 weeks (range 52 – 69 weeks). The number of scheduled and completed sessions and compliance to the scheduled training for each quarter of the programme (0-3, 3-6, 6-9 and 9-12 months) are given in Tables 2.4 and 2.5. The average number of sessions completed was 186.8 out of the 232 scheduled sessions giving an average compliance of 81 %. Compliance was highest at 0-3 months (86 %) and was steady thereafter at 79-80 %.

Subject	1	2	3	4	5	Average	Total scheduled
0-3 Month	45	42	28	39	34	**37.6**	**44.0**
3-6 Month	49	46	44	45	61	**49.0**	**62.0**
6-9 Month	61	50	39	40	59	**49.8**	**63.0**
9-12 Month	61	46	44	48	53	**50.4**	**63.0**
Total	**216**	**184**	**155**	**172**	**207**	**186.8**	**232.0**

TABLE 2.4: NUMBER OF SESSIONS COMPLETED.

Subject	1	2	3	4	5	Average
0-3 Month	102.3	95.5	63.6	88.6	77. 3	**85.5**
3-6 Month	79.0	74.2	71.0	72.6	98.4	**79.0**
6-9 Month	96.8	79.4	61.9	63.5	93.7	**79.0**
9-12 Month	96.8	73.0	69.8	76.2	84.1	**80.0**
Average	**93.7**	**80.5**	**66.6**	**75.2**	**88.4**	**80.9**

TABLE 2.5: COMPLIANCE WITH TRAINING PROGRAMME.

As shown in Table 2.6, the most common reasons for missing training were unknown, skin and equipment problems. 'Unknown' is given where subjects failed to report reasons for missing training. Examples of the skin problems experienced by subjects are shown in Fig. 2.3.

Reason	0-3 Month	3-6 Month	6-9 Month	9-12 Month	Total
Holiday	0	7	9	6	**22**
Work	0	0	5	7	**12**
UTI	0	1	3	3	**7**
Unwell	0	1	4	4	**9**
Equipment failure	9	9	0	8	**26**
Skin	0	11	7	8	**26**
Injury	0	4	2	1	**7**
Unknown	22	31	38	37	**128**

TABLE 2.6: REASONS FOR SESSIONS MISSED, UTI = URINARY TRACT INFECTION.

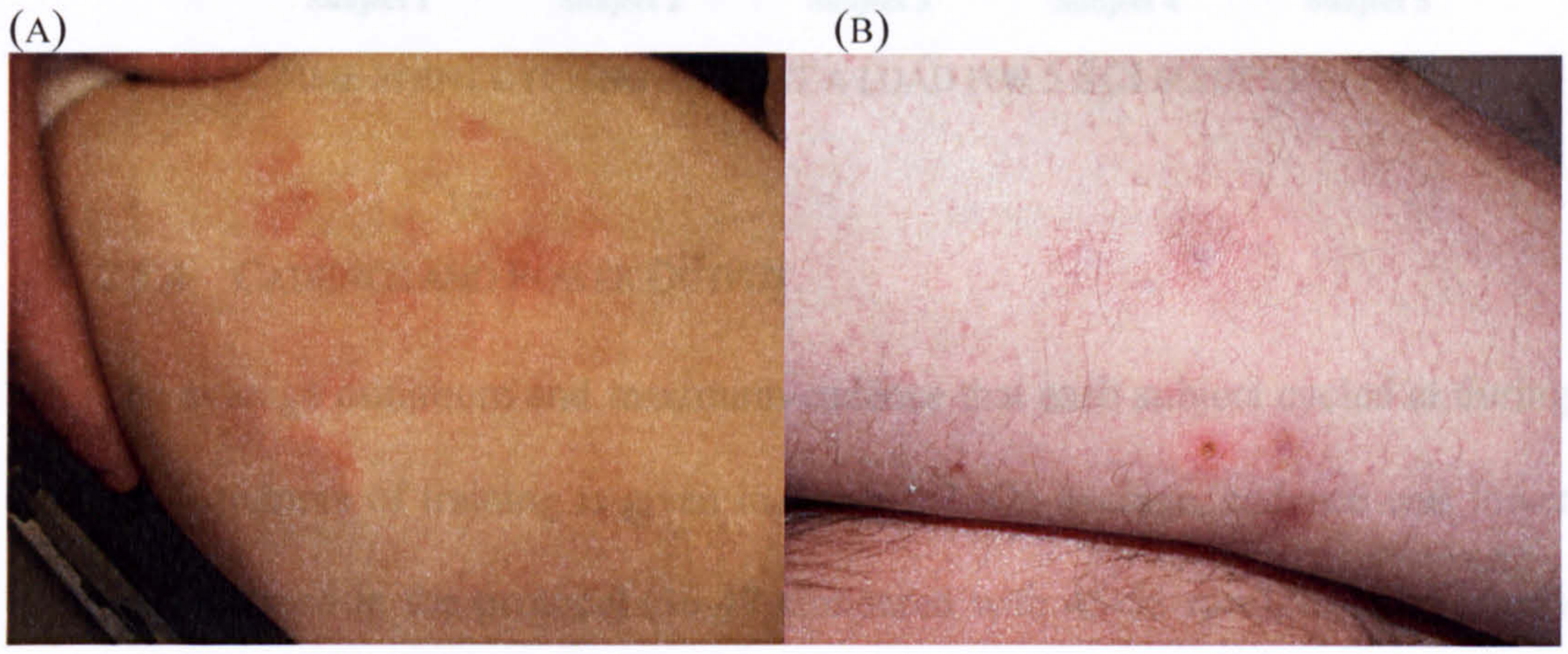

FIG. 2.3: EXAMPLES OF SKIN PROBLEMS EXPERIENCED BY SUBJECTS DURING TRAINING (A) AS THEY FIRST APPEARED, IMMEDIATELY POST-TRAINING AND (B) IN THE LATTER STAGES.

2.5.3 TIME SPENT CYCLING WITH LOAD

The amount of time each subject spent cycling against a load during each quarter is shown in Fig. 2.4. There was a substantial increase in training time for all 5 subjects during 3-6, 6-9 and 9-12 months compared with 0-3 months. Subjects 1 and 2 gradually increased their training time throughout the programme, whereas the other 3 subjects decreased training time after 6 or 9 months.

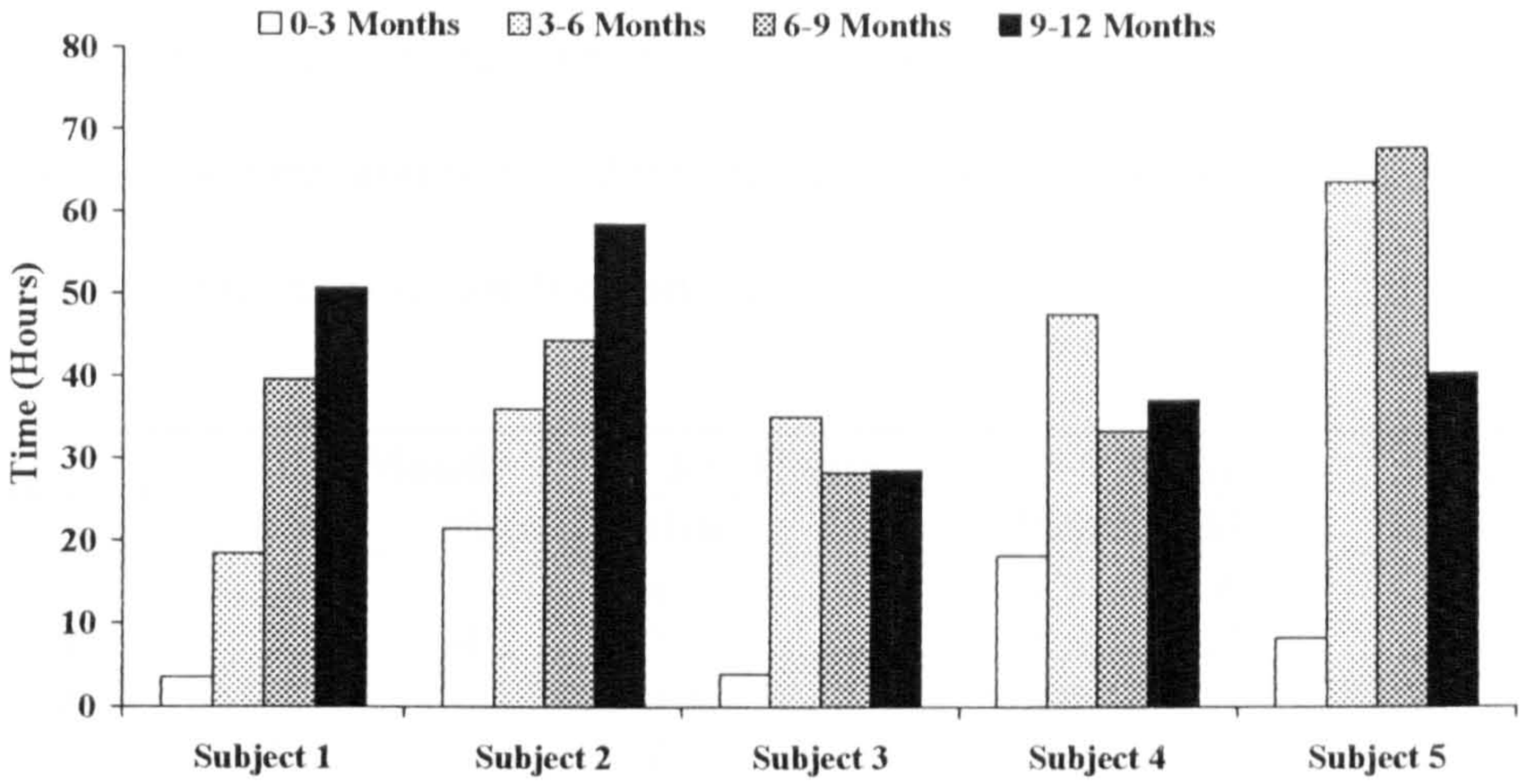

FIG. 2.4: TIME SPENT CYCLING AGAINST A LOAD FOR 5 SCI SUBJECTS.

2.5.4 CADENCE AND POWER OUTPUT

The average minimum and maximum cadence that each subject cycled at during the 4 quarters of training is given in Table 2.7. On average, cadence was lower than outlined in the protocol (36-47 compared with 45-55 rpm), but was highly variable across subjects (range 25-50 rpm). During each one hour training session cadence tended to decrease. For most subjects, cadence gradually increased throughout the one year programme (Table 2.7).

Subject	0-3 Month		3-6 Month		6-9 Month		9-12 Month	
	Min	Max	Min	Max	Min	Max	Min	Max
1	--	--	27.1	31.8	37.4	46.3	41.6	43.0
2	44.1	45.2	41.0	46.2	45.9	50.3	44.7	49.1
3	--	--	33.0	47.2	24.7	50.5	24.5	45.1
4	37.0	44.4	41.8	44.7	43.1	45.1	42.0	45.3
5	36.8	37.6	38.7	40.0	38.6	40.9	37.3	39.1
Average	**39.3**	**42.4**	**36.3**	**42.0**	**37.9**	**46.6**	**38.0**	**44.3**

TABLE 2.7: AVERAGE MINIMUM AND MAXIMUM CADANCE (REVOLUTIONS PER MINUTE) DURING EACH SESSION (-- DATA NOT REPORTED).

Average power output during home training is given in Table 2.8. This ranged from 4.0-5.0 W at the start of training to 6.5-8.4 W at the end of training. Some data points are missing from cadence and power data due to equipment faults and failure of subjects to report data consistently.

Subject	0-3 Month		3-6 Month		6-9 Month		9-12 Month	
	Min	Max	Min	Max	Min	Max	Min	Max
1	--	--	4.5	6.8	4.9	8.9	6.3*	6.5*
2	3.5	4.2	3.7	5.1	8.5	10.3	5.7	7.5
3	--	--	3.6	7.0	2.4	6.5	2.8	7.1
4	3.6	5.9	6.3	7.0	11.3	12.4	11.1	12.4
5	4.8*	5.0*	6.0*	7.0*	--	--	--	--
Average	**4.0**	**5.0**	**4.8**	**6.6**	**6.8**	**9.5**	**6.5**	**8.4**

TABLE 2.8: AVERAGE MINIMUM AND MAXIMUM POWER OUTPUT DURING EACH SESSION (-- DATA NOT REPORTED, * SOME DATA NOT REPORTED).

2.6 DISCUSSION

2.6.1 COMPLIANCE

Average compliance for all subjects over the one year programme was 80.9 %. As shown in Table 5 compliance decreased after the first three months. Presumably this occurred because training frequency was lowest during the initial three months (3-4 sessions per week). Compliance during this period (85.5 %) is comparable to the 90 % compliance previously reported for FES cycling programmes being carried out three times per week (Faghri et al., 1992; Sköld et al., 2002). There was an additional 5-6 % decrease in compliance during the final 3-12 months, when training frequency was increased. To my knowledge, compliance with a training programme involving 5 sessions per week has not previously been reported for FES cycling. The subjects with the highest overall compliance were those not in full-time work (Subjects 1 and 5). It should be noted that each session takes approximately 2 hours including set up time and

therefore it is understandable that people with commitments such as full time work would be unable to maintain such a regimen.

Four out of the 5 subjects experienced skin problems under the electrodes (Fig. 2.3) during the training period. As can be seen in Table 6, skin problems began during the 3-6 month period where training increased from 4 to 5 sessions per week. When skin problems occurred, subjects missed sessions to allow their skin to recover, causing compliance to decrease. After this, subjects who suffered skin problems reduced the training frequency to prevent the occurrence of further skin problems. Reducing the number of sessions per week to 3-4 sessions prevented the recurrence of skin problems indicating that the high frequency of training sessions contributed to these problems. The cause of these skin problems is unclear. Subjects might have developed an allergy to the electrodes (although hypoallergenic electrodes were used) or high temperature or current density under electrodes might be causative factors.

Equipment problems were greatest at the start of training. As subjects became more familiar with the equipment problems occurred less frequently. Other reasons for missing training (holiday, illness) were reported by all subjects as expected during a one year period. Additionally, urinary tract infections (UTI's) are common for people with SCI and were reported by two subjects as reasons for missing training.

2.6.2 TIME SPENT CYCLING WITH LOAD

All subjects increased their time cycling against a load from 0-3 to 3-6 months, as required by the training programme. Two subjects continued to increase their training time throughout the programme. The other 3 subjects began to decrease their training time after 6 or 9 months. It is likely that the reduced compliance after 6 months contributed to this. Furthermore, the substantial skin problems experienced by these subjects meant that their training time after 6 months was reduced to prevent any recurrence of these problems.

It is also likely that subjects became de-motivated towards the latter stages of the training programme. Peak power output and maximal isometric strength began to plateau for some subjects at 9 and 12 months as shown in chapters 3 and 4, respectively. A plateau in training is likely to be de-motivating, particularly with such a long-term and intense training regimen.

2.6.3 CADENCE

As shown in Table 2.7, only subjects 2 and 4 were able to maintain a cadence of 45-50 rpm. At the start of training the other subjects found that they were unable to cycle at 50 rpm with resistance at the lowest trainer setting (-4). Therefore, in order for training to progress these subjects were asked to cycle at 35-45 as opposed to 45-55 rpm as originally outlined in the protocol. They were then able to gradually increase the resistance, maintaining their cadence at a lower level. These cadences are similar to those previously reported for FES cycling, with studies reporting a cadence of 50 decreasing to 35 rpm with fatigue (Arnold et al., 1992; Faghri et al., 1992; Hooker et al., 1992; Sköld et al., 2002)

At the start of training the three subjects unable to cycle at 45-55 rpm (1, 3 and 5) were also the weakest subjects (assessed by maximal isometric quadriceps force, see Chapter 3). Maximal force achieved by Subject 5 improved substantially through out the year (4.8 and 151.2 Newton-Metres (Nm) at baseline and 12 months respectively, see Chapter 3). This subject became able to cycle at 50 rpm at all trainer settings, but continued training at 35-45 rpm with higher resistance. Subjects 1 and 3 remained weakest (maximal force = 47.2 and 91.3 Nm at 12 months, see Chapter 3) and did not develop enough strength over one year to cycle at 50 rpm. Nonetheless their maximal force did increase throughout the programme (see Chapter 3) as did their cadence, indicating a positive relationship between cadence and muscle strength.

2.6.4 POWER OUTPUT

Power output gradually increased over time for all subjects from 4-5 W after 3 months to 7-10 W after 9 months of training (Table 2.8). Power output decreased during the final quarter (7-8 W), which might be because of the concurrent decrease in compliance (Table 2.5). These values are generally lower than power outputs previously reported of 0-55 W following a short-term FES training programme (Pacy et al., 1987; Ragnarrson et al., 1988; Arnold et al., 1992; Faghri et al., 1992; Hooker et al., 1992; Sköld et al., 2002). However, these studies did report large variability between subjects, with some subjects training at 0 W after 36 sessions (Ragnarsson et al., 1988). It should also be noted that these studies used a more upright seating position, which might have contributed

these discrepancies. The differences in cycling ability during recumbent compared with upright FES cycling requires further investigation.

Power output data reported here should be interpreted with caution due to missing data values and low subject numbers. Furthermore, it was not possible to monitor power output for the duration of each session so these values provide only a guideline. It is likely that error of measurement for the ergotrainer occurred because it was not possible to calibrate the trainer according to the manufacturer's guidelines as this requires the individual to cycle at a speed of 30km.hour^{-1}. Additionally, the tyre pressure of the trike is likely to change over time, which would affect the tyre diameter and thus the amount of resistance applied by the ergotrainer. In order to gain accurate power output data it would be necessary to attach a force transducer to the crankshaft of the front pedal, and monitor power output through out the session. This technique was used during laboratory tests (at baseline, 3, 6, 9 and 12 months, see Chapter 4) to gain precise power output data.

2.7 CONCLUSIONS

All subjects were willing and able to complete a one year programme of FES cycling. However, training 5 times per week appeared not to be practical for people in full-time work. Skin problems occurred when training at this frequency and it is suggested that training 3-4 times per week should be the maximum.

Two out of 5 subjects were able to cycle at the required cadence (45-55 rpm) through out the training programme. The remaining subjects were unable to train

at this cadence, presumably due to low muscle strength. Both cadence and power output data collected during the training programme should be interpreted with caution due to limitations involved with subjective data collection techniques and inaccuracies with the equipment used.

Time spent cycling with load was increased substantially for all subjects after the initial 3 months training. After 6 or 9 months training, training time reduced for some subjects in line with a reduction in training compliance. This is probably due to skin problems and reduced motivation.

The following chapter reviews current literature concerning muscular adaptations in response to ES training and FES cycling in SCI people. Muscle adaptations as a result of the present FES cycling programme are also presented and discussed.

Chapter 3 Muscle Properties.

This chapter investigated changes in muscle size strength and contractile properties and also subcutaneous tissue thickness over a one year FES cycle training programme.

3.1 Literature Review

3.1.1 Electrical Stimulation

Electrical stimulation (ES) can be applied cutaneously to elicit muscular contraction by generating the depolarisation of intramuscular motor nerves. Increasing both pulse height and width will initially generate increased force by recruiting a greater population of motor units and muscle fibres.

The activation of a muscle group by ES differs from a voluntary contraction in two main ways:

- ES elicits synchronous firing from all active muscle fibres as opposed to the asynchronus firing that occurs in voluntary contraction.
- The normal hierarchical pattern of recruitment is altered. With ES there is a nonselective and synchronous recruitment of motor units (Gregory & Bickel, 2005).

ES has been shown to have short-term peripheral effects equivalent to voluntary muscle activity in able-bodied people (Arnold et al. 1992). Similar training adaptations have been identified after voluntary endurance training and long-term low frequency stimulation both in the early (one week) and latter (6 weeks) stages of a training programme (Salmons & Henriksson, 1981).

3.1.2 MUSCLE SIZE AND STRENGTH

A number of studies have noted increases in muscle cross sectional area (CSA) following a short (Petrofsky, 1987; Arnold et al., 1992; Hjeltnes et al., 1997; Mohr et al., 1997; Scremin et al., 1999; Crameri et al. 2002; Sköld et al., 2002) or long (Pacy et al., 1988; Mohr et al. 1997) term programme of FES cycling in SCI people. Pacy et al. (1987) noted a significant increase in the number of internal nuclei of muscle fibres following a period of FES cycle training in SCI people and suggested this could be due to continued muscle damage.

Increases in muscle strength have also been identified following ES training in both AB (Cabric & Appell, 1987a; 1987b) and SCI people (Rochester et al., 1995a; Crameri et al., 2002; Gerrits et al., 2002), which is probably due to increases in muscle size, although SCI people remain significantly weaker compared with AB (Rochester et al., 1995). Pacy et al. (1987) noted that the significant increases in quadriceps area of both legs appeared unrelated to the power outputs attained by paraplegics during FES cycling.

Low frequency (≤20 Hz) ES (LFES) training has been shown to result in reduced force production in both animal (Gordon et al., 1997) and human (Rutherford & Jones 1988) intact muscle. This is probably due to a fast-slow fibre type transformation resulting in a reduced fibre size, calcium release and contractile ability of the muscle. (Salmons & Henriksson, 1981). Similarly, little or no strength gain in SCI people has been reported following LFES (Martin et al.,

1992), which is probably due to the lack of mechanical or metabolic stimulus for increases in muscle size and therefore strength.

It has been reported that no changes in adipose tissue thickness occur following a programme of FES cycling (Scremin et al., 1999; Sköld et al., 2002). However, significant increases in the muscle mass:adipose tissue ratio have been noted (Hjeltnes et al., 1997; Scremin et al., 1999).

3.1.3 Fibre Type Transformation

It has long been established that prolonged LFES in normal muscle alters the physiological, morphological and contractile properties of fast muscle towards those of slow muscle (Salmons & Sreter, 1976; Salmons & Henriksson, 1981; Eisenberg & Salmons, 1981; Hudlicka et al., 1982; Eerbeek et al., 1984). A leftward shift in the force:frequency curve following chronic LFES has also been noted in intact human adductor pollicis muscle (Edwards et al., 1982; Rutherford & Jones, 1988).

In SCI people, a programme of ES has been shown to induce significant increases in the percentage of type IIa fibres and a significant decrease in the percentage of IIx fibres compared with pre-training values (Greve et al., 1993; Crameri et al., 2002). FES cycle training has also been shown to induce a switch in the myosin heavy chain (MHC) composition of single fibres from predominantly containing only MHC IIx to predominantly containing only MHC IIa after a one year training programme (Anderson et al., 1996).

SCI people have been reported to show a significantly greater proportion of type II fibres compared with AB people and a very small proportion of fibres containing MHC I both pre- and post- ES training (Martin et al., 1992; Anderson et al., 1996) even after one year of training (Anderson et al., 1996). However, some studies have reported increases in the proportion of type I fibres following an ES training programme (Martin et al., 1992; Crameri et al., 2002), although these increases were small and the proportion of type I fibres remained well below that of AB people (Martin et al., 1992). Large variability in the proportion of type I fibres in untrained paralysed muscle has been reported (Martin et al., 1992; Gerrits et al., 2003) with some SCI people showing a complete lack of type I fibres and others a more normal distribution (Pacy et al., 1987). This might explain the discrepancies in type I fibre changes with ES training between studies. Harridge et al. (2002) reported no significant effects of a 4 week ES training programme on MHC isoform distribution, however there was a significant improvement in the number of fibres that contained the mRNA MHC I transcript indicating that to some extent a fast-slow fibre type conversion would eventually occur. The extent of fibre-type transformation is probably dependent upon the frequency, intensity and duration of ES.

The activities of both oxidative (citrate synthase and 3-hydroxyacyl-CoA dehydrogenase) and glycolytic (hexokinase and lactate dehydrogenase) enzymes have been shown to increase following a short-term programme of FES cycling in SCI people (Kjear et al., 2001a; Crameri et al., 2002) but this has been reported to level off after 3 months (Kjær et al., 2001a).

Slowing of contractile speed has also been reported following a period of ES training in SCI people (Stein et al., 1992; Rochester et al., 1995b). However, Gerrits et al. (2000a) reported a righward shift in the force:frequency relationship at low frequencies, indicating a faster contractile speed, following a 6 week programme of FES cycling. This study also reported decreases in twitch half relaxation time following the FES training, which also contradicts a fast to slow fibre type transformation. It was suggested that the re-uptake of calcium by the SR is impaired in fast muscles of SCI people due to long periods of inactivity. This might improve with training, resulting in the observed decrease in relaxation times (Gerrits et al., 2000a). It is also possible that the frequency and duration of training used in that study (30 minutes, 3 times per week) was too low to induce slowing of contractile speed. Indeed, Stein et al. (1992) reported no change in contractile speed following ES training for 15 or 45 minutes per day whereas ES training >2 hours per day resulted in significant slowing.

It has been suggested that contractile slowing is dependent upon the duration of stimulation (Kernell & Eerbeek, 1989) and number of impulses (Stein et al. 1992; Sutherland et al. 1998; Lopez-Guajardo et al., 2001) per session. Wide variations in half relaxation time have been reported, with both faster and slower times reported in untrained paralysed muscle compared with muscles of AB people (Rochester et al., 1995a), which might also explain these discrepancies.

3.1.4 FATIGUE RESISTANCE

Animal studies have shown very early adaptations to chronic LFES including increased activity of oxidative enzymes and a concomitant increase in

mitochondrial density and Z-line thickness (Eisenberg & Salmons, 1981) resulting in improved fatigue resistance (Eerbeek et al., 1984; Kernell et al., 1988).

In paralysed muscle, improved fatigue resistance following ES training has been well established (Stein et al. 1992; Rochester et al., 1995b; Gerrits et al. 2000a; Gerrits et al., 2002). This has been attributed to increased capillary density and mitochondrial content (Salmons & Henriksson, 1981) and significant increases in the activity of glycolytic and oxidative enzymes (Rochester 1995; Kjær et al. 2001; Crameri et al., 2002). It also consistent with a transformation of type IIx to IIa muscle fibres (Martin et al., 1992; Greve et al., 1993; Anderson et al., 1996; Crameri et al., 2002).

3.1.4.1 Blood Flow

Following LFES endurance training an adaptive increase in the capillary:fibre ratio has been shown in both intact animals (Salmons & Henriksson, 1981; Hudlicka, 1989) and able-bodied individuals (Cabric et al., 1987c; Nash et al., 1996). This may occur as early as 4 days following LFES (Brown et al., 1976).

Similar results have also been shown following a period of ES training in SCI people (Chilibeck et al., 1999; Crameri et al., 2002). A period of FES cycling has been shown to reverse the vascular atrophy and reduced lower limb blood flow that occurs due to reduced activity in paralysed limbs (Hopman et al., 1994a; 1996; Olive et al., 2003a). Gerrits et al. (2001a) reported a significant increase in the cross sectional area of the common femoral artery, increased blood inflow

volume and reduced peripheral resistance at rest following 6 weeks of FES cycling. Furthermore, the number of capillaries surrounding each muscle fibre has been reported to increase significantly following 10 weeks of FES training (Crameri et al., 2002). These adaptations are likely to contribute to the improved cycling performance by enhancing oxygen delivery and removal of metabolic products. It is also possible that these adaptations will improve the microcirculation in paralysed parts leading to improved tissue health and a reduced susceptibility to pressure sores.

3.1.5 PATTERN OF STIMULATION

Previous studies investigating the effects of ES on paralysed muscle have used a variety of stimulation patterns. In order to design an effective, convenient and safe training programme, a stimulation pattern needs to be used that does not induce rapid muscular fatigue, permits improvements in muscle strength and is not too time-consuming for SCI individuals. The design and validation of such programmes limits the current use of FES cycling.

3.1.5.1 Pulse amplitude

Glaser et al. (1994) compared the effects of stimulation at 130 and 300 milliamperes (mA) during a graded FES cycling exercise test. 300 mA resulted in a significant increase in peak power of 124 %, although the absolute values were not reported. It also resulted in a significantly higher peak oxygen uptake and blood lactate concentration at peak power.

3.1.5.2 Pulse Frequency and Duration

The force:frequency relationship of muscle shows greater improvements in force when frequency is increased at lower pulse frequencies (<100 Hz) but little or no increase in force when pulse frequency is increased above 100 Hz. The frequency at which a fused tetanus occurs is indicative of the speed of a muscle. With fast muscle there is less fusion at low frequencies resulting in relatively less force at low frequencies and a rightward shift of the force:frequency relationship. With slower muscle, greater fusion and therefore force is generated at low frequencies causing the force:frequency relationship to shift rightwards.

Reductions in muscle strength with LFES in AB people (Kernell et al., 1987; Gordon et al., 1997) appear to be less pronounced when training with higher pulse frequencies (Hudlicka et al., 1982; Eerbeek et al., 1984). Kernell & Eerbeek (1989) identified muscle fibre diameter to be better preserved following training with high frequencies of identical duration. In SCI people however, studies have shown training at high and low frequencies to have similar effects on improvements in muscle strength (Cabric et al., 1987a; Gerrits et al., 2002).

Studies investigating the long-term effects of ES have noted contractile slowing and improved fatigue resistance in intact animal (Hudlicka et al., 1982; Eerbeek, et al., 1984; Kernell et al., 1987; Gordon et al., 1997) and paralysed human (Stein et al., 1992; Rochester et al., 1995a; Harridge et al., 2002) muscle irrespective of pulse frequency. Kernell & Eerbeek (1989) found changes in twitch speed to be highly dependent upon the duration of stimulation per session, as opposed to pulse frequency. It has since been suggested that the aggregate number of pulses

per session is the important factor to bring about contractile slowing and fatigue resistance (Stein et al. 1992; Sutherland et al. 1998; Lopez-Guajardo et al., 2001).

Eser et al. (2003) reported that 60Hz stimulation during FES cycling did not induce greater fatigue than 30Hz. Higher frequency trains (>33 Hz) have been reported to produce more repetitive knee contractions that met a 50° target excursion than lower frequency trains (Kebaetse et al., 2002; Kebaetse & Binder Macleod, 2004). In order to preserve muscle strength and endurance, higher frequency stimulation patterns that produce greater tension appear to be more effective (Kebaetse et al., 2002; Sutherland et al., 2003). For the practical use of FES cycling, maintenance of muscle strength as well as fatigue resistance is important.

3.1.5.3 Intermittent Frequencies and Variable Frequency Trains

An alternative method that offsets the reduction in muscular strength noted with LFES, without inducing rapid fatigue, incorporates short periods of high frequency stimulation interspersed on a low frequency background (Kernell et al. 1987). In normal human muscle, Rutherford & Jones (1988) compared a uniform 10Hz stimulation pattern with a non-uniform pattern containing high frequency bursts against an essentially low frequency background. They found a decrease in force production with the uniform pattern only.

The use of these variable frequency trains (VFT's) incorporated into constant frequency trains appear to create greater force production and offset fatigue

(Russ & Binder-MacLeod, 1999; Scott & Binder-MacLeod, 2003). VFT's commonly involve the use of a doublet or triplet at the onset of stimulation. This aims to utilise the catchlike property of skeletal muscle (Burke et al., 1970; Ding et al., 2003). It has been shown however that VFT's and constant frequency trains (CFT's) have similar effects during high frequency stimulation (Kebaetse & Binder-Macleod, 2004). Bickel et al. (2003) also reported VFT's and CFT's at approximately 14Hz to have similar effects on peak torque and rate of contractions in non-fatigued muscle. In fatigued muscle however, VFT's have been shown to evoke greater peak torque and less slowing of rise time than CFT's (Binder-Macleod et al., 1997; Bickel et al., 2003). Thus VFT's might be useful during ES at the onset of fatigue.

In normally innervated human muscle the firing rates of fatiguing motor units decrease with exercise duration, and simultaneously new motor units are recruited which begin firing at high frequencies (Carpentier et al., 2001). The synchronous firing that occurs during ES means that such an effect cannot be replicated. Nonetheless, it has been reported that low followed by high frequency stimulation produces a higher number of joint movements during repetitive knee extension exercise compared with low and high frequency CFT's, high frequency VFT's and high followed by low CFT's (Kebaetse & Binder-Macleod, 2004). This suggests that in fatigued muscle the use of higher frequency stimulation or VFT's offsets fatigue by improving peak torque and reducing the reduction in rise time that occurs. The time at which stimulation frequency should be altered remains unclear. Furthermore, the inter-subject variability noted during ES at different frequencies (Kebaetse et al., 2002;

Kebaetse & Binder Macleod, 2004) indicates that there is no single optimal stimulation pattern for all individuals (Kebaeste & Binder-Macleod, 2004).

3.1.5.4 Effect of Load

Studies that have incorporated resistance into a programme of electrical stimulation have found substantially greater muscle strength gains (Petrofsky, 1987; Scremin et al., 1999; Bélanger et al., 2000; Hartkopp et al., 2003; Crameri et al., 2004). Crameri et al. (2004) noted greater increases in isometric force, fibre CSA and capillary:fibre ratio in human muscle trained isometrically rather than dynamically. Since cycling at lower cadences (15 rpm) has been shown to produce higher peak crank torque's than when cycling at 50 rpm (Fornusek & Davis, 2004) it is possible that cycling at a lower cadence would be favoured for improvements in strength due to the force:velocity relationship of muscle.

Improvements in aerobic metabolism also have been reported following resisted training (Hartkopp et al., 2003). Petrofsky & Laymon (2004) noted a substantial improvement in endurance capability during FES cycling as a consequence of concurrent weight training.

3.1.5.5 Training frequency and duration

Petrofsky (1987) reported increases of limb girth of SCI people of 1.82, 2.70 and 4.33 cm following 4 weeks of ES induced isokinetic weight training completed 1, 3 or 5 times per week, respectively. However, Scremin et al. (1999) reported that muscle hypertrophy was unrelated to the number of FES cycling sessions carried

out. This discrepancy might be because isokinetic weight training provided greater resistance than cycling.

Contractile slowing and improvements in fatigue resistance have been reported to be dependant on the duration of stimulation (Kernell & Eerbeek, 1989) or the aggregate number of stimulation pulses (Stein et al. 1992; Sutherland et al. 1998; Lopez-Guajardo et al., 2001) per session. This indicates that high frequency, long duration sessions might be optimal for improvements in both muscle size and fatigue resistance.

This chapter reports muscle size, strength and contractile properties before and at three monthly intervals throughout a one year period of FES training in SCI people. Their were compared with a group of AB people.

3.2 METHODOLOGY

A one year FES cycling programme was carried out at a stimulation frequency of 50 Hz, incorporating 1 hour training per day, 5 days per week was completed by the 5 SCI people, as described previously (Chapter 2). Ten AB people (5 female) mean age, height and weight 30.6 ± 3.2 years, 172.6 ± 1.9 cm and 69.5 ± 3.1 kg, respectively, were also tested on one occasion.

3.2.1 MUSCLE AND SUBCUTANEOUS TISSUE SIZE

At Guy's Hospital, MRI scans were taken of the lower limbs at baseline and following 12 months of training. Subjects were positioned prone in an MRI

scanner (Phillips, Acheiva 1.5T (Release 1.5) using the body coil). A number of slices were taken to identify the proximal border of patella and the tibial tubicle. Transverse slices of the right and left thigh were taken 15 cm above the proximal border of the patella. Transverse slices of the right and left calf were taken 10cm distal to the tibial tubicle. Cross-sectional area of muscle groups, bone and subcutaneous tissue were measured using custom designed software (Matlab programming; Woledge, 2006, unpublished) for left and right thigh and calf slices. The software measures CSA by finding the area of a polygon in pixels. The area was defined using a series of dots and the line between two dots was interpolated by the software (Appendix 6). Three measurements of each muscle were taken to ensure accuracy.

Ultrasound images were taken using an Aloko SSD-900 (Aloko Co Ltd, Tokyo, Japan) with a 7.5 MHz linear transducer. Images were taken of the left quadriceps and calf muscles at all time-points for the 5 SCI subjects and of the left quadriceps in 5 AB people. Quadriceps muscle depth was measured on the midline of the anterior thigh, 15 cm above the proximal border of patella. Calf muscle depth was measured in the midline of the posterior calf, 10 cm distal from the tibial tuberosity. Subjects were laying supine (quadriceps) or prone (calf) on a couch. The area of skin was cleaned with an alcohol swab and covered with conductive gel. Muscles were scanned in the transverse plan ensuring the transducer was vertical and pressing lightly on the skin to ensure the image was not distorted. A vertical line was then drawn on each image from the femur (quadriceps) or tibia (calf) to the surface of the skin using in built software. Measurements of muscle thickness were taken between the fascia boundaries and

subcutaneous tissue depth measurements were taken from the outer fascia boundary to the surface of the skin. These measurements were also made from MRI images of the right quadriceps to ensure validity between the two measurement techniques.

3.2.2 CONTRACTILE PROPERTIES

Muscle contractile properties were assessed to determine changes in muscle strength and fatigue resistance and also to indicate fibre type composition. All muscle tests were carried out with electrical stimulation applied to the left quadriceps muscle through two electrodes (Physio Med, approximately 12 x 12 cm) placed as far apart as possible on the anterior-lateral aspect of thigh, bandaged to place (Fig. 3.1). Five SCI people were tested at three monthly intervals throughout the FES training programme and 10 AB people were tested on one occasion.

Subjects were positioned on an isokinetic dynanomemter (Kin-Com, Chattecx Corporation, USA) (calibrated regularly) so that the seat of the testing chair supported the whole of their thigh and the ankle was strapped to the lever arm of the dynanmometer with the knee set at 90° flexion (Fig. 3.1). The system was set to isometric mode with a sampling rate of 750 Hz. A stimulator (Digitimer Stimulator DS7, England) and pulse generator (Digitimer D4030, England) applied stimulation with pulse width of 200 μs and square wave-form.

Analogue force signal were collected and digitised (CED 1401) and recorded on a personal computer. Data was analysed offline using signal software.

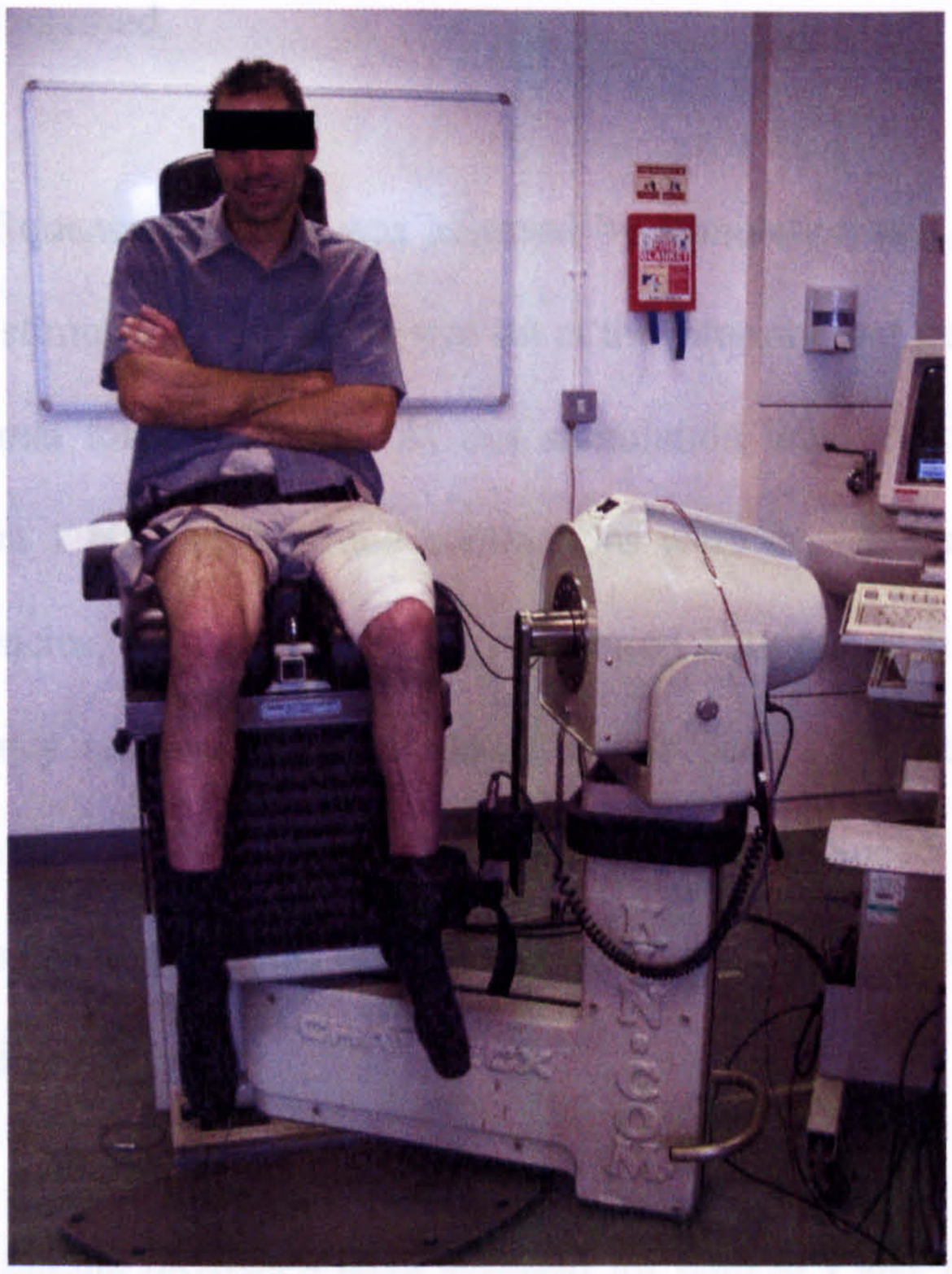

FIG. 3.1: SUBJECT POSITIONED IN ISOKINETIC DYNANMOMETER WITH LEFT KNEE AT 90° FLEXION AND ELECTRODES BANDAGED TO LEFT THIGH.

For maximal force in SCI people, stimulation intensity at 50 Hz was increased gradually to elicit isometric contractions. The maximal force was taken when the force produced ceased to increase with increased stimulation intensity (amplitude) or declined due to the antagonists also being stimulated. The maximal force (in N from calibration) and the lever arm length were recorded to calculate torque in Nm. If torque attained was >50 Nm, stimulation frequency was reduced to 20 Hz and the maximum force was extrapolated from the 20/50 Hz relationship in order to reduce the risk of bone fracture. AB people were assessed for maximum force by performing maximum voluntary contractions

(MVC) separated by 10 second rest periods until force produced ceased to increase or decreased.

The torque:frquency response was assessed by stimulation at 1, 10, 20, 50 and 100Hz. The stimulation amplitude was set at the intensity that generated 20-30% of the maximal force at 50 Hz. At this stimulation intensity, 5 twitches were carried out at 1 Hz and 2-second contractions were carried out at each of the other frequencies, with a two second rest period between each frequency. The force:frequency response was calculated by the peak force generated at each frequency, expressed as a percentage of that at 100 Hz. Speed of relaxation (at 100 Hz) was calculated as the time taken for force to fall from 80 to 45% of the force created at 100 Hz.

Changes in fatigue resistance were assessed for both SCI and AB people using the modified Burke fatigue protocol (Burke et al., 1974). Frequency was set at 40 Hz and stimulation intensity as above. The muscle was stimulated for 250 ms per second for 180 seconds. The percentage force loss at 1, 2 and 3 minutes were calculated from the initial force.

3.2.3 Data Analysis

Analysis of variance was carried out on all muscle size, strength, torque:frequency response and fatigue resistance data for SCI people through out the one year training programme. Post-hoc analysis was carried out using paired Student's T-Tests. Data collected on AB people was compared with SCI people at all time points using unpaired Student's T-tests.

3.3 RESULTS

Maximal torque, torque:frequency, twitch time and fatigability data is based on n=4 at 6 months only due to technical problems with subject 4.

3.3.1 MUSCLE AND SUBCUTANEOUS TISSUE SIZE

Quadriceps muscle thickness (assessed by ultrasound) increased gradually throughout the training programme (Fig. 3.2) and had changed significantly at 3, 6, 9 and 12 months compared with baseline ($P < 0.01$). At baseline and 3 months quadriceps muscle thickness was significantly less for SCI compared with AB people ($P < 0.01$ and < 0.05, respectively) but similar at 6, 9 and 12 months. Subcutaneous tissue thickness at the anterior thigh tended to decrease throughout the training programme (Fig. 3.2) but this did not attain statistical significance. Subcutaneous tissue thickness at the thigh was similar for SCI and AB people at all time points.

Muscle thickness at the posterior calf increased gradually until 6 months into the training programme and reached a plateau thereafter (Fig. 3.3). Calf muscle thickness was significantly greater at 3 ($P < 0.05$), 6, 9 and 12 ($P < 0.01$) months than baseline. Similarly, subcutaneous tissue at the posterior calf decreased until 6 months into the training and reached a plateau thereafter, however this did not attain statistical significance ($P > 0.05$).

Quadriceps muscle thickness had improved by 76, 88, 110 and 118 % at 3, 6, 9 and 12 months respectively. Calf muscle thickness improved to a lesser extent and reached a plateau after 6 months (22, 31, 32 and 31 % at 3, 6, 9 and 12 months).

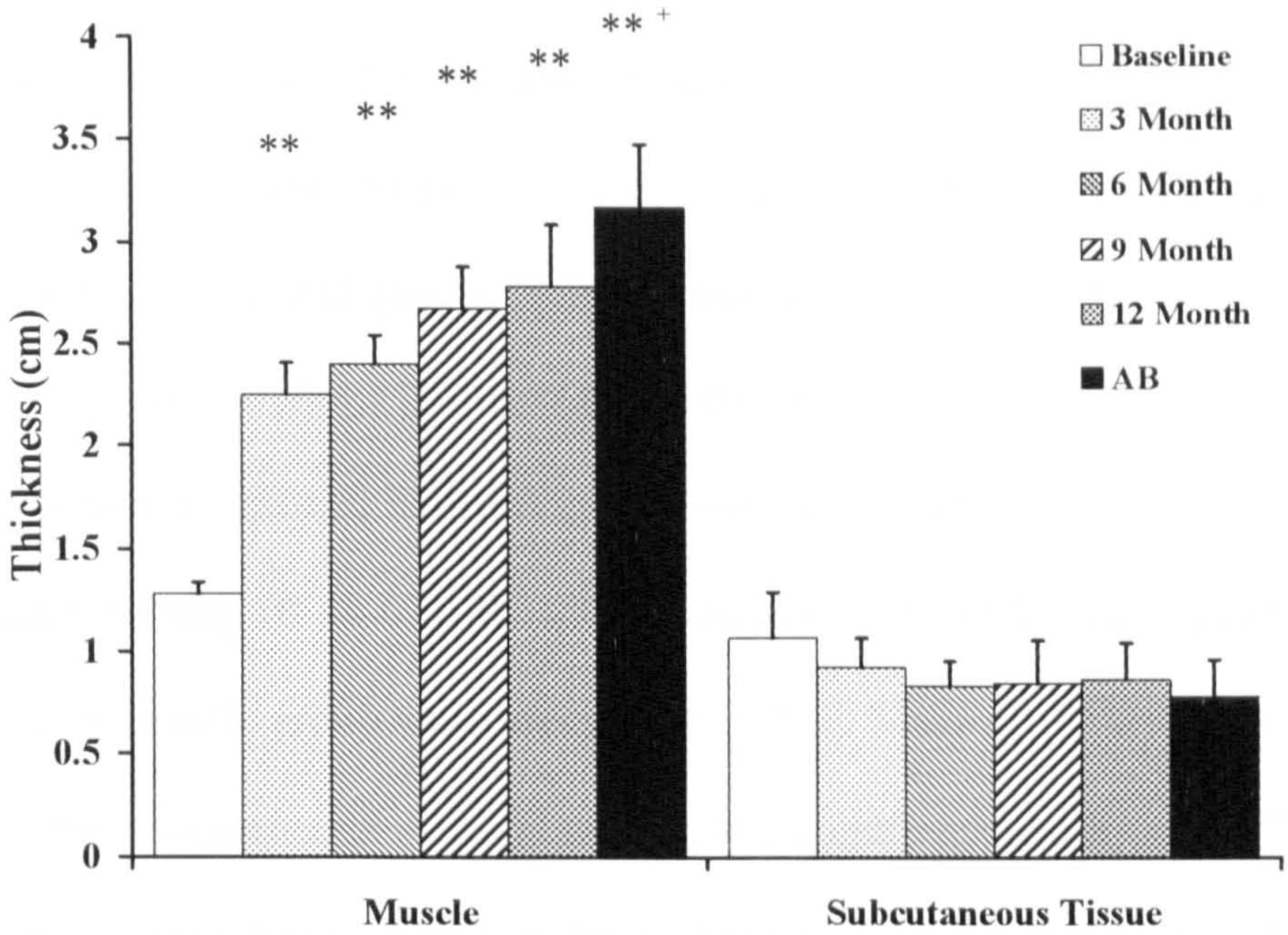

FIG. 3.2: ANTERIOR THIGH MUSCLE AND SUBCUTANEOUS TISSUE THICKNESS (MEAN AND SEM) FOR SCI SUBJECTS AT THROUGHOUT THE TRAINING PROGRAMME AND FOR AB PEOPLE (SIGNIFICANTLY DIFFERENT TO BASELINE ** $P < 0.01$, SIGNIFICANTLY DIFFERENT TO 3 MONTH $^{+}$ ($P < 0.05$)).

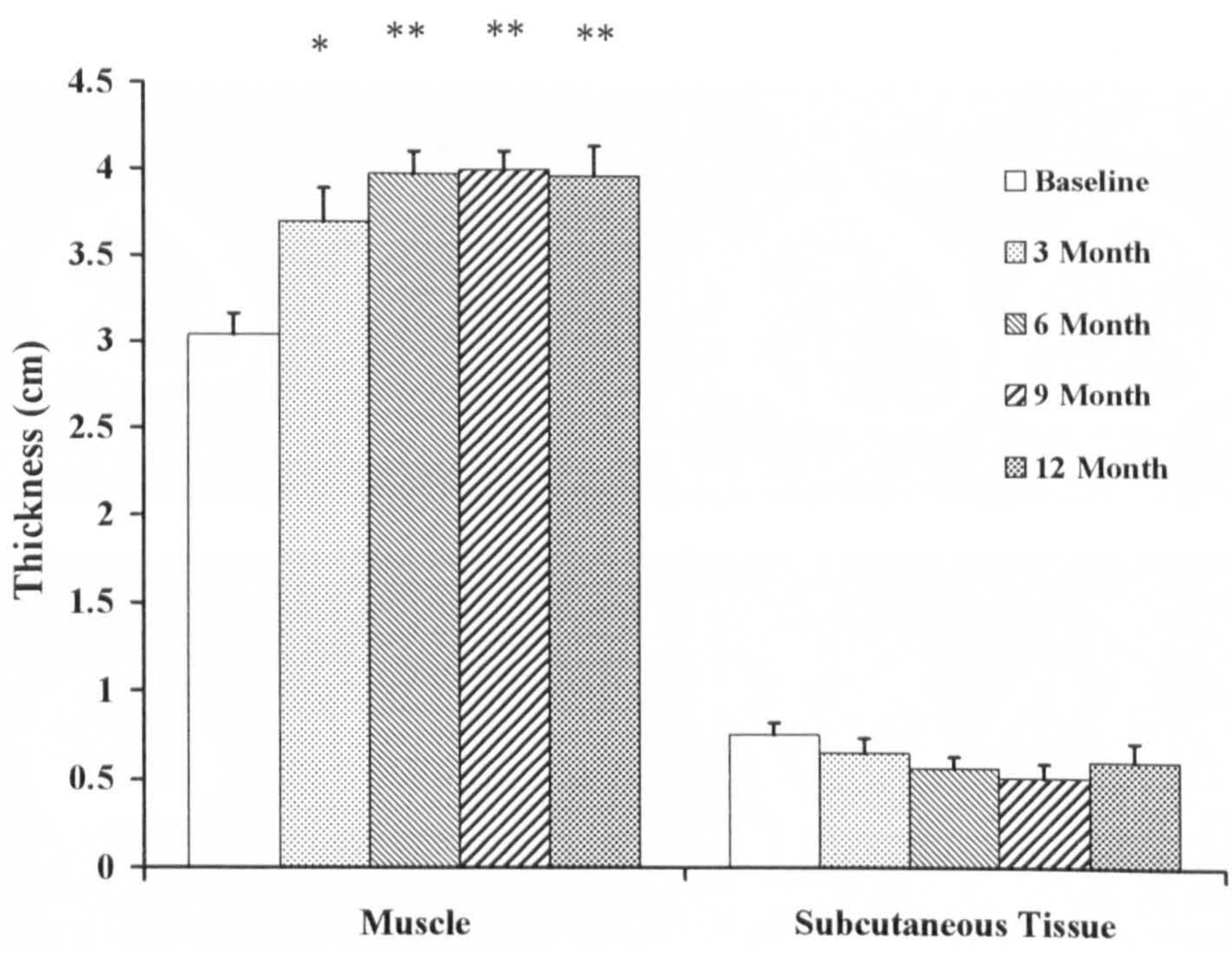

FIG. 3.3: POSTERIOR CALF MUSCLE AND SUBCUTANEOUS TISSUE THICKNESS (MEAN AND SEM) FOR SCI SUBJECTS THROUGHOUT THE TRAINING PROGRAMME (SIGNIFICANTLY DIFFERENT TO BASELINE * ($P < 0.05$), ** ($P < 0.01$)).

An example of MRI images taken at the thigh and calf before and after training is shown in Fig. 3.4. CV for repeated measures were 2.7 and 4.0 % for the thigh and calf respectively. Mean (± SEM) cross sectional area of the quadriceps, hamstrings, gluteals and gastrocnemius/soleus increased by 54.9 ± 9.5, 30.7 ± 3.4, 30.7 ± 13.7 and 31.9 ± 11.0 %, respectively, after 12 months training (Fig. 3.5). Only the quadriceps and hamstrings showed significant improvements in muscle size after training ($P < 0.01$). Relative increases in the individual muscles of the quadriceps muscle group were variable (Fig. 3.6). Mean (± SEM) cross sectional area of the subcutaneous tissue at the thigh and calf changed by –7.4 (7.7) and -9.5 (3.6) % respectively, however these changes were not statistically significant (Fig 3.7). Muscle mass:adipose tissue ratio however increased significantly in the thigh ($P < 0.01$) and calf ($p < 0.05$).

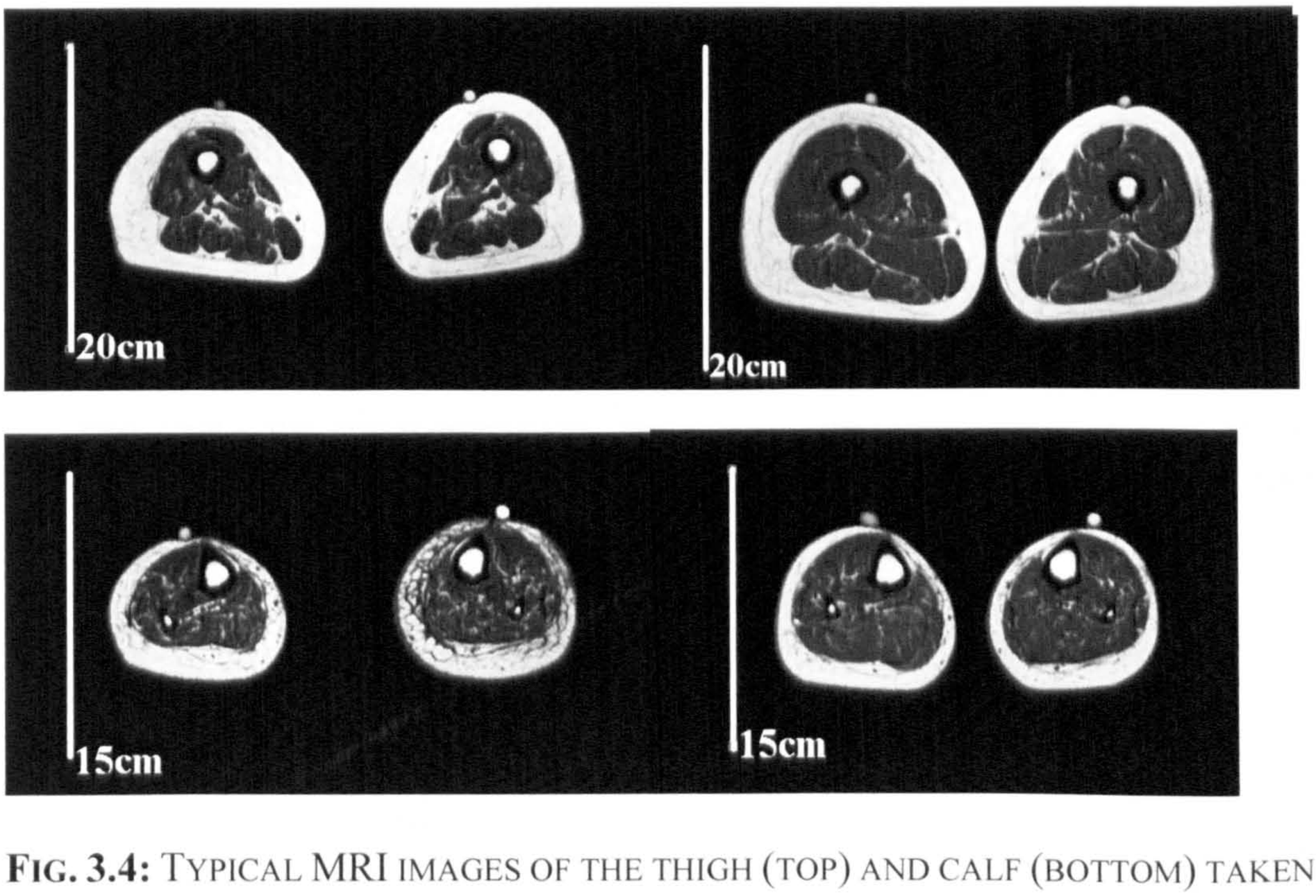

FIG. 3.4: TYPICAL MRI IMAGES OF THE THIGH (TOP) AND CALF (BOTTOM) TAKEN BEFORE (LEFT) AND AFTER (RIGHT) TRAINING IN ONE SUBJECT.

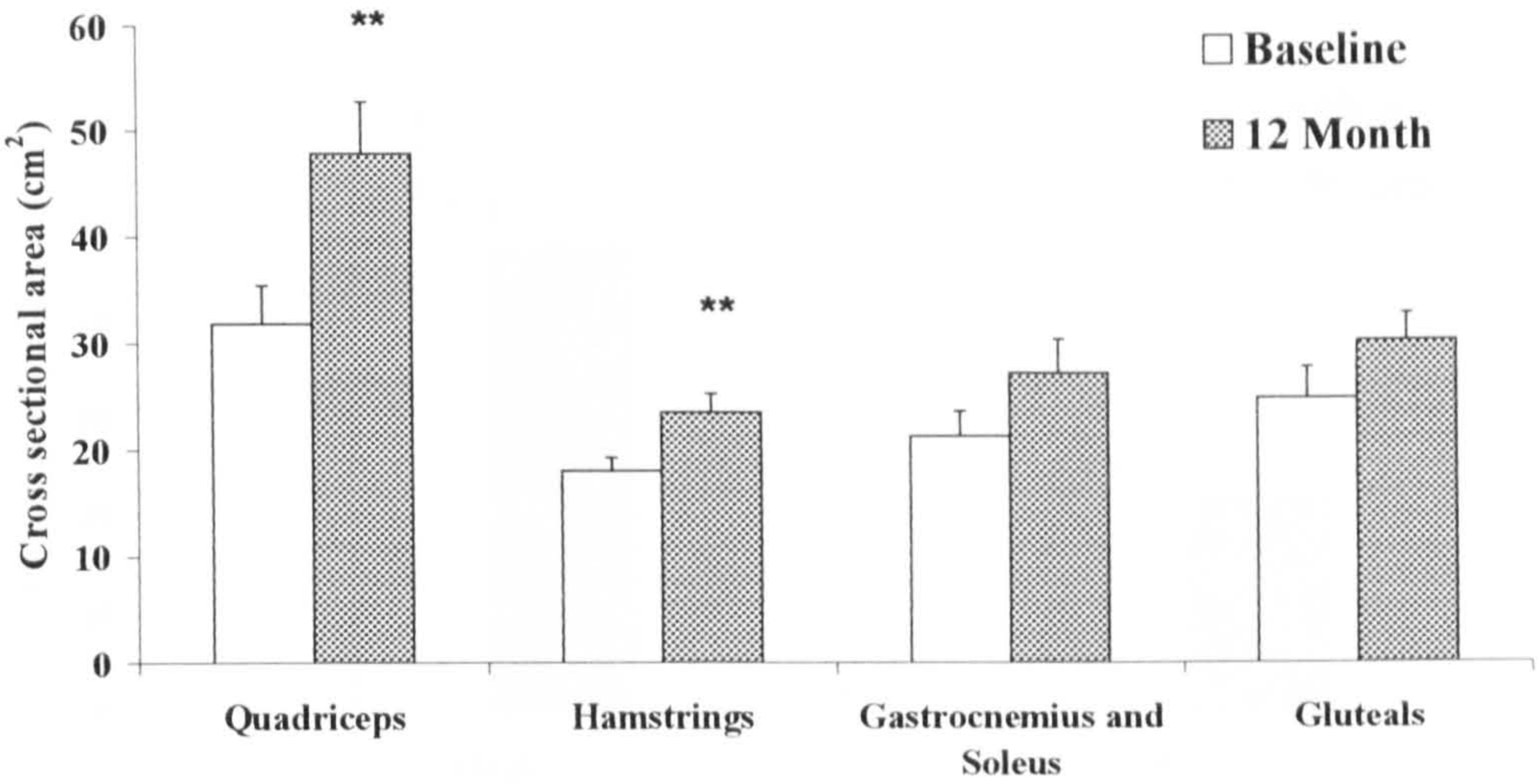

FIG. 3.5: MEAN (± SEM) CROSS SECTIONAL AREA OF QUADRICEPS, HAMSTRINGS, GASTROCNEMIUS/SOLEUS AND GLUTEAL MUSCLE AT BASELINE AND AFTER 12 MONTHS TRAINING (** SIGNIFICANTLY DIFFERENT TO BASELINE, $P < 0.01$).

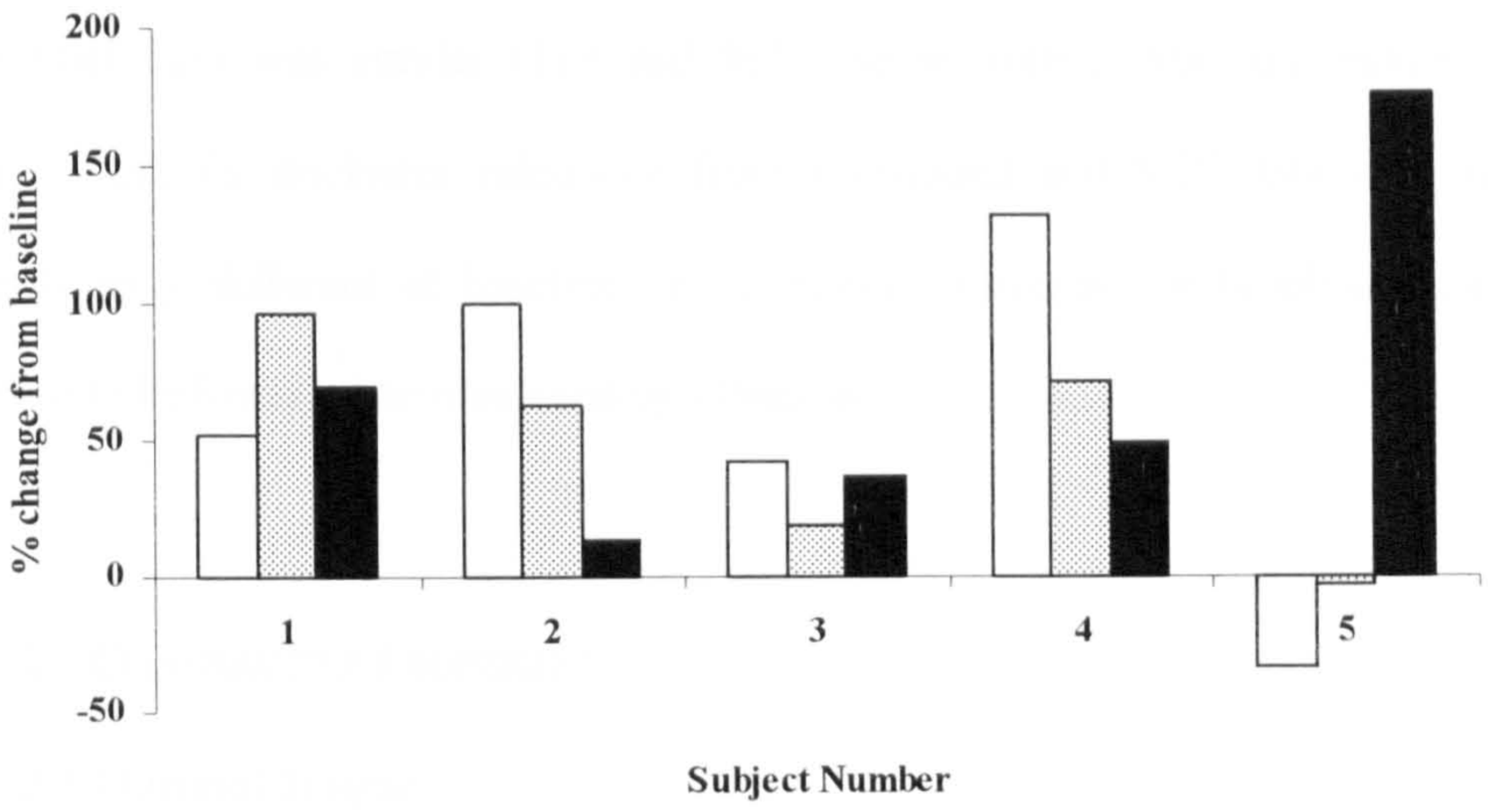

FIG: 3.6: RELATIVE INCREASE IN RECTUS FEMORIS (OPEN BARS), VASTUS INTERMADILUS AND LATERALIS (GREY BARS) AND VASTUS MEDIALIS (BLACK BARS) AFTER TRAINING FOR EACH SUBJECT.

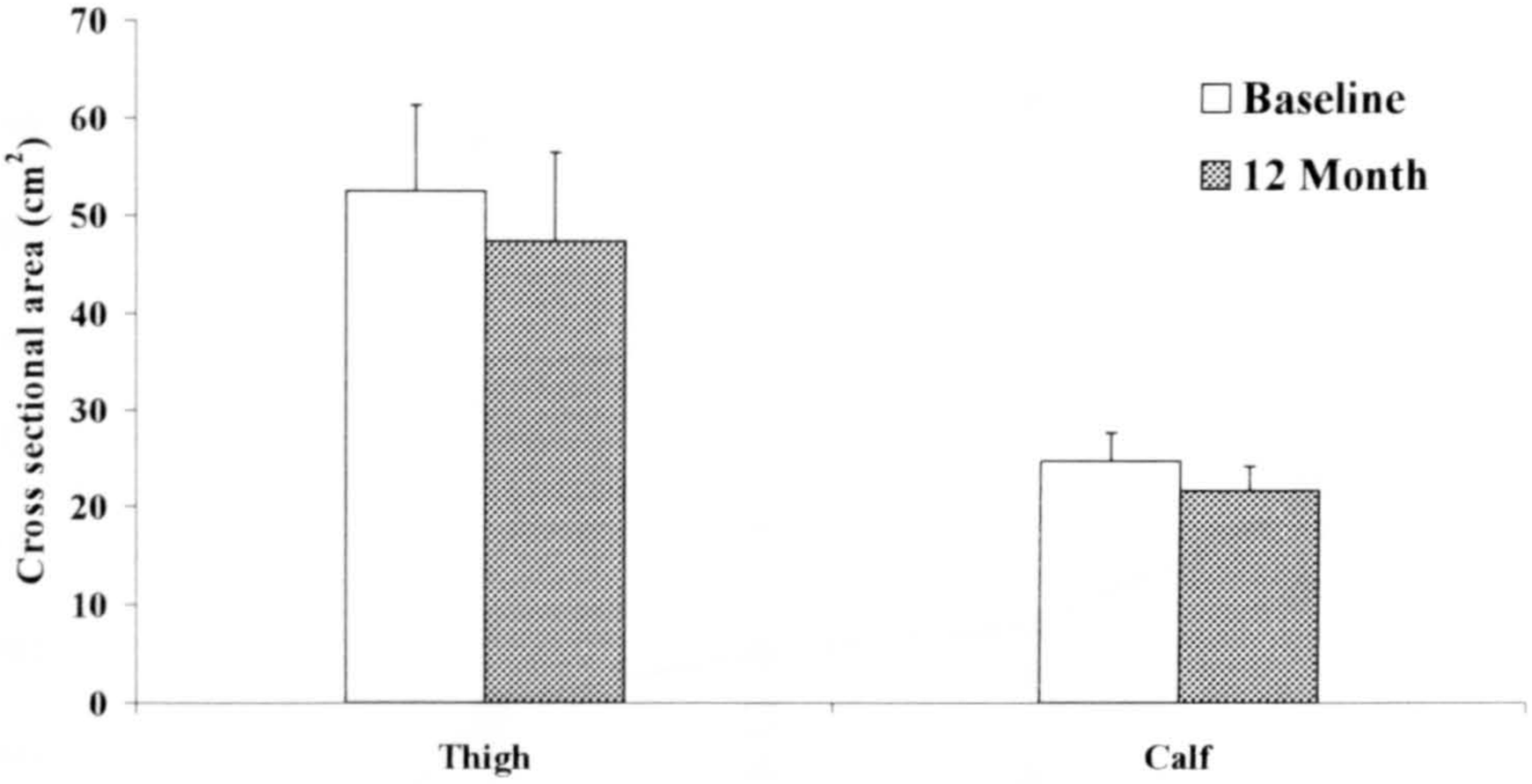

FIG. 3.7: MEAN (± SEM) CROSS SECTIONAL AREA OF SUBCUTANEOUS TISSUE AT THE THIGH AND CALF AT BASELINE AND AFTER 12 MONTHS TRAINING.

The improvement in quadriceps muscle depth measured by ultrasound and from the MRI data was similar (118 and 91%, respectively). Absolute values of muscle and fat thickness calculated from ultrasound and MRI data were not significantly different at baseline or 12 month, however the baseline values tended to be lower when measured by ultrasound.

3.3.2 CONTRACTILE PROPERTIES

3.3.2.1 Maximal Torque

ES elicited maximum torque of the quadriceps increased substantially at 3 months compared with baseline and continued to increase gradually thereafter (Fig 3.8).

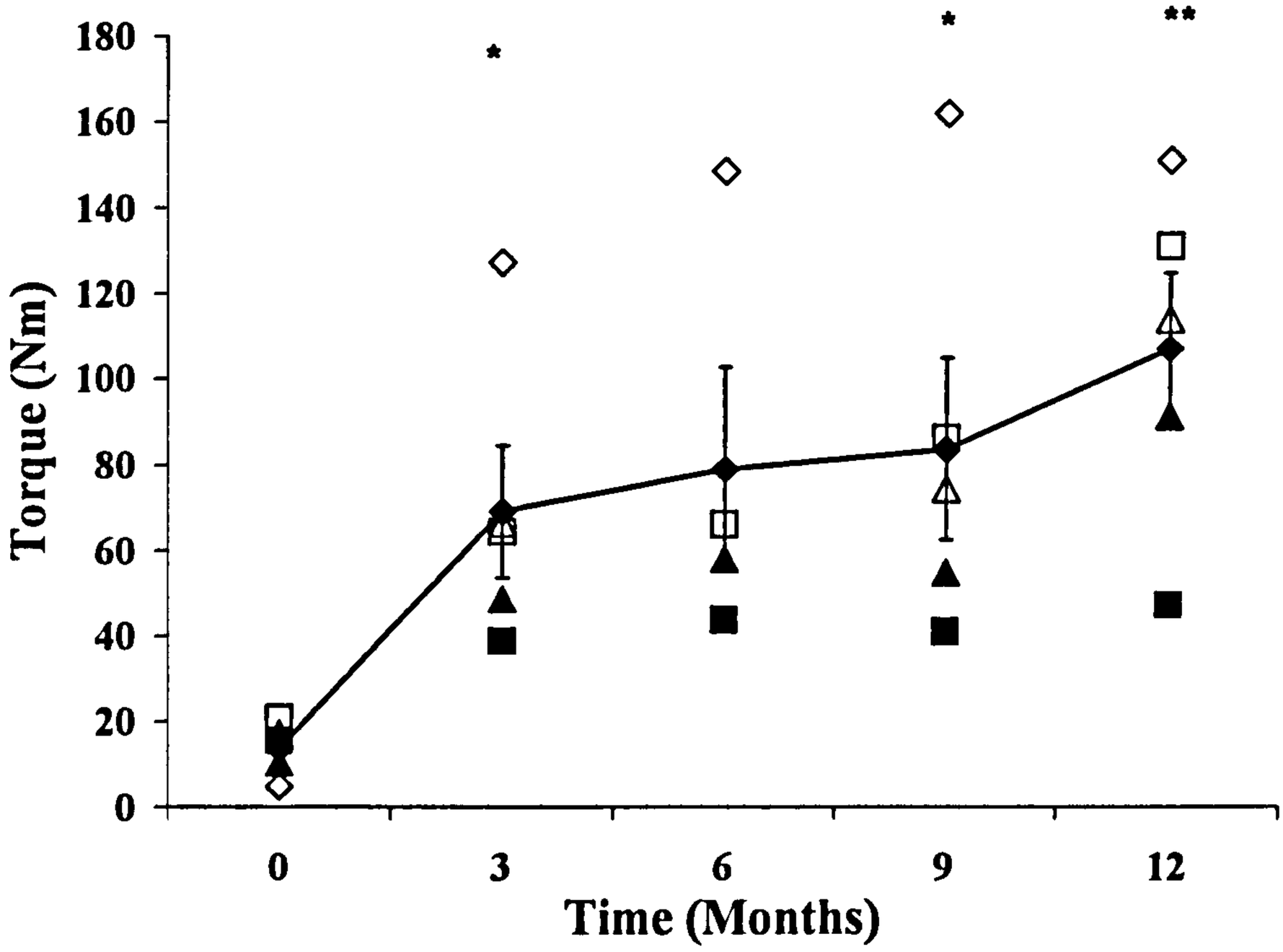

FIG. 3.8: MAXIMUM TORQUE (CLOSED DIAMOND AND LINE, MEAN AND SEM) AND INDIVIDUAL DATA (SUBJECT 1 CLOSED SQUARE, 2 OPEN SQUARE, 3 CLOSED TRIANGLE, 4 OPEN TRIANGLE AND 5 OPEN DIAMOND) OVER THE YEAR OF TRAINING. (SIGNIFICANTLY DIFFERENT TO BASELINE * ($P < 0.05$), ** ($P < 0.01$).

Maximum torque had increased significantly at 3, 9 ($P < 0.05$) and 12 ($P < 0.01$) months compared with baseline, however remained significantly lower than the MVC's of the untrained AB people (Fig. 3.9). Average ES elicited maximum force in SCI people was 4.2, 20.9, 23.9, 25.3 and 32.3 % of MVC's in AB people at baseline, 3, 6, 9 and 12 months.

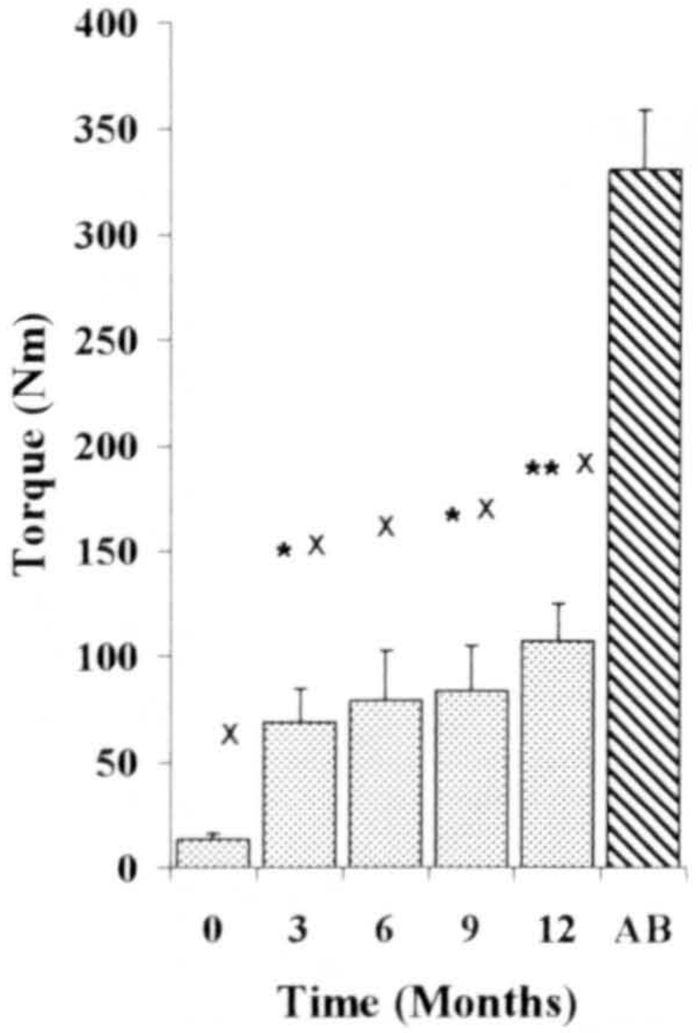

FIG 3.9: AVERAGE ES ELICITED MAXIMUM TORQUE FOR SCI PEOPLE OVER THE YEAR OF TRAINING (GREY BARS) AND AVERAGE MVC FOR 10 UNTRAINED AB PEOPLE (BLACK AND WHITE BAR) (SIGNIFICANTLY DIFFERENT TO BASELINE *($P < 0.05$), **($P < 0.01$), SIGNIFICANTLY DIFFERENT TO AB ^{X} ($P < 0.01$)).

For SCI people, changes in quadriceps muscle thickness correlated significantly with changes in muscle strength (R^2 = 77.0 %, $P < 0.01$, Fig. 3.10). Fig. 3.11 shows maximum torque and quadriceps muscle thickness for 5 AB people and the 5 SCI people after one year training. ES elicited maximum torque in SCI people appeared to be lower than expected based on quadriceps muscle thickness when compared with AB people.

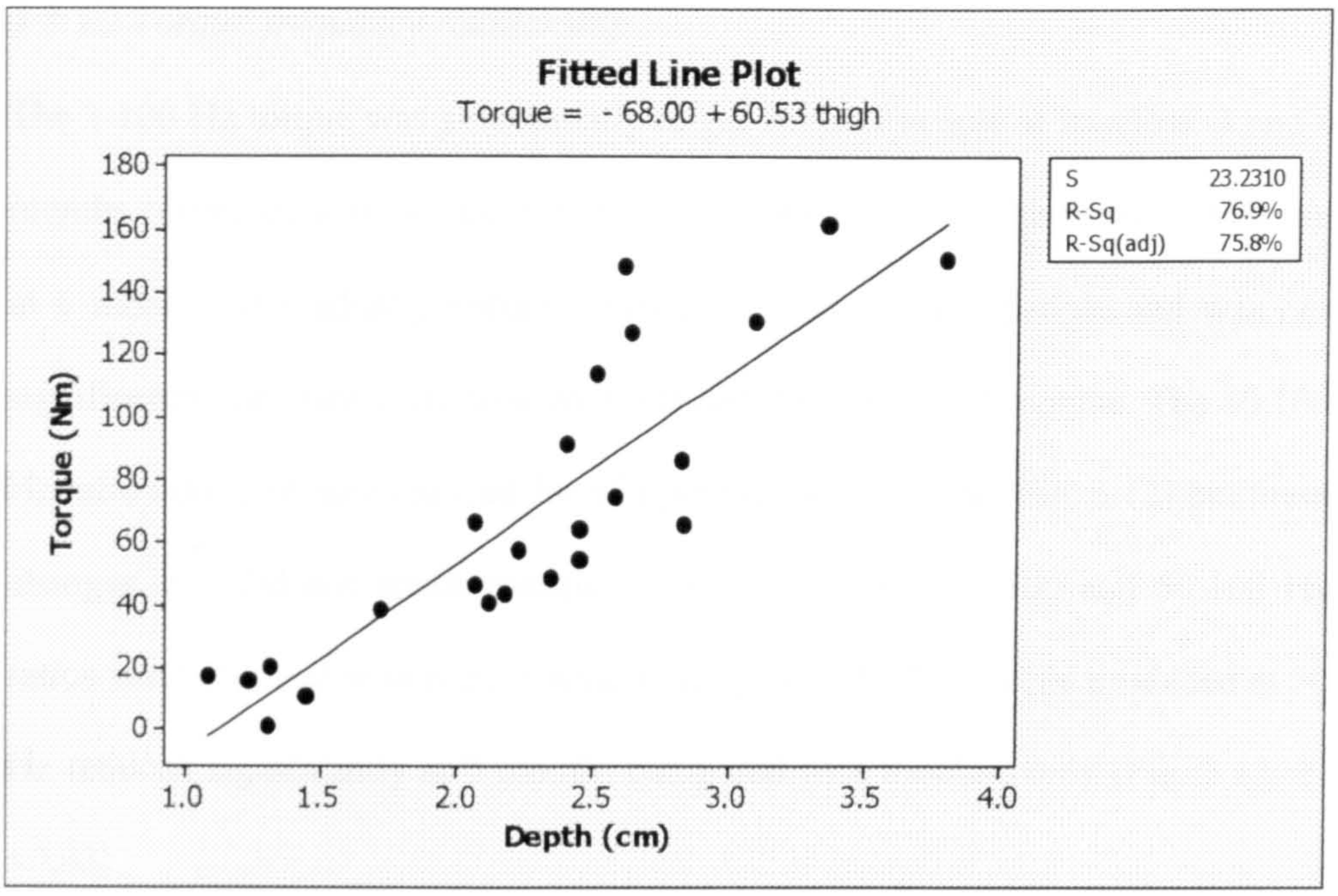

Fig 3.10: QUADRICEPS MUSCLE DEPTH AND MAXIMAL TORQUE FOR 5 SCI PEOPLE THROUGHOUT THE TRAINING PROGRAMME.

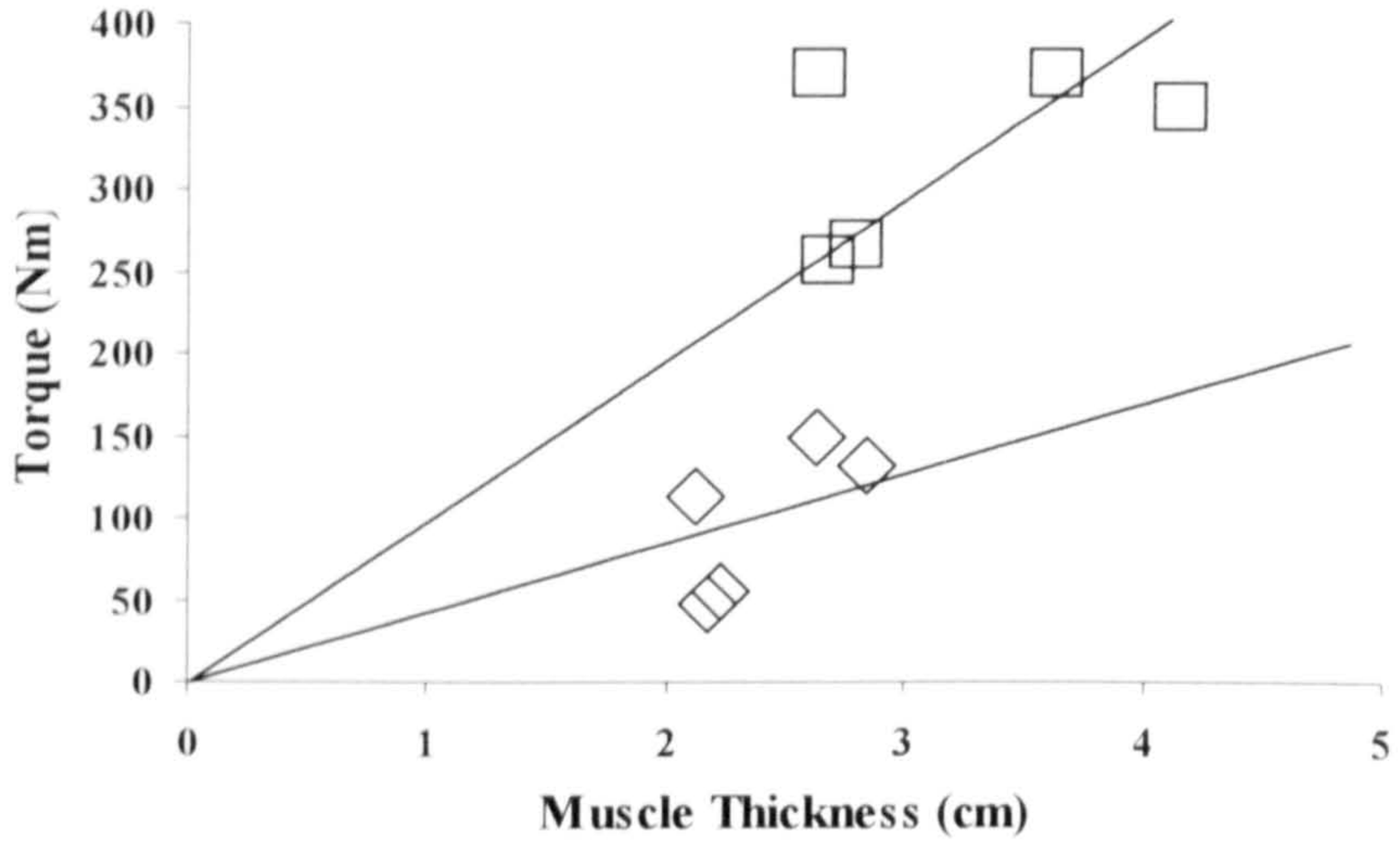

Fig 3.11: QUADRICEPS MUSCLE DEPTH AND MAXIMAL TORQUE FOR 5 SCI PEOPLE (OPEN DIAMONDS) FOLLOWING ONE YEAR TRAINING AND 5 UNTRAINED AB PEOPLE (OPEN SQUARES).

3.3.2.2 Torque:frequency relationship

The 1:100 Hz torque was substantially larger for SCI people at baseline, 3 and 6 months compared with AB people (Fig. 3.12) but was only significantly different at 6 months. It gradually reduced during training for SCI people and was not significantly different compared with AB people at 9 and 12 months. The 10:100 Hz ratio also gradually reduced for SCI people over the year (Fig. 3.12) but these changes also did not attain statistical significance. The 20:100 and 50:100 Hz ratios similarly tended to reduce with training (Fig. 3.12). Torque produced at 50 Hz reduced significantly at 9 months compared with baseline only (Fig. 3.12, P. <0.05).

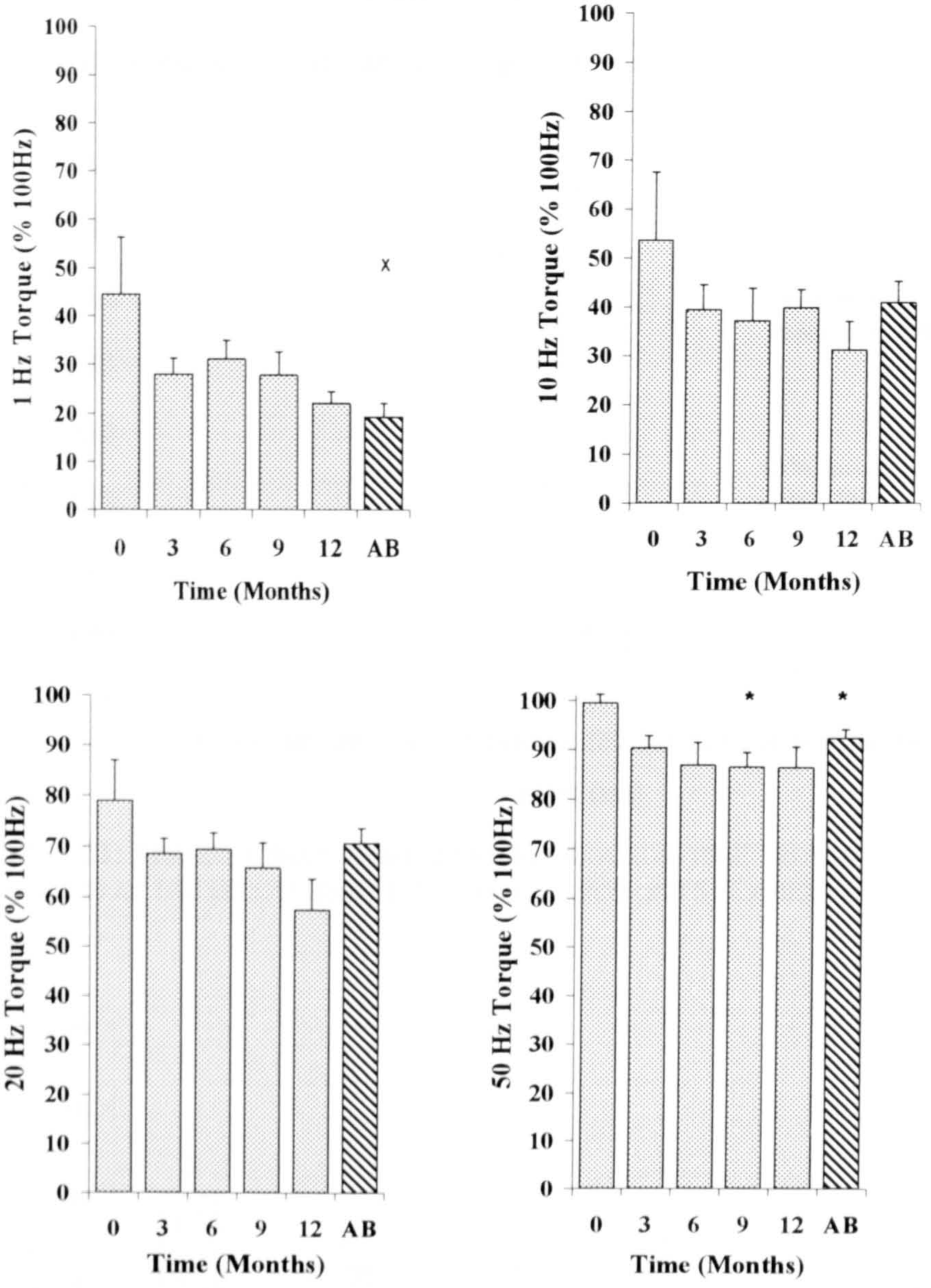

FIG 3.12: TORQUE AT 1, 10, 20 AND 50 HZ NORMALISED TO THAT AT 100 HZ (MEAN AND SEM) FOR SCI PEOPLE OVER THE YEAR OF TRAINING (GREY BARS) AND FOR 10 UNTRAINED AB PEOPLE (BLACK AND WHITE BAR) (SIGNIFICANTLY DIFFERENT TO BASELINE * ($P < 0.05$).

Figure 3.13 shows the raw data trace from 100 Hz tetani in one SCI person and the method used to measure relaxation times. Time taken for torque created at 100 Hz to reduce from 80-45 % was 40.4 (7.2) ms at baseline, which was slower than untrained AB people (30.9 (2.1) ms). In SCI people relaxation time tended

to reduce with training, although not significantly, and was more comparable with AB people after 12 months training (32.0 (1.6) ms, Fig. 3.14).

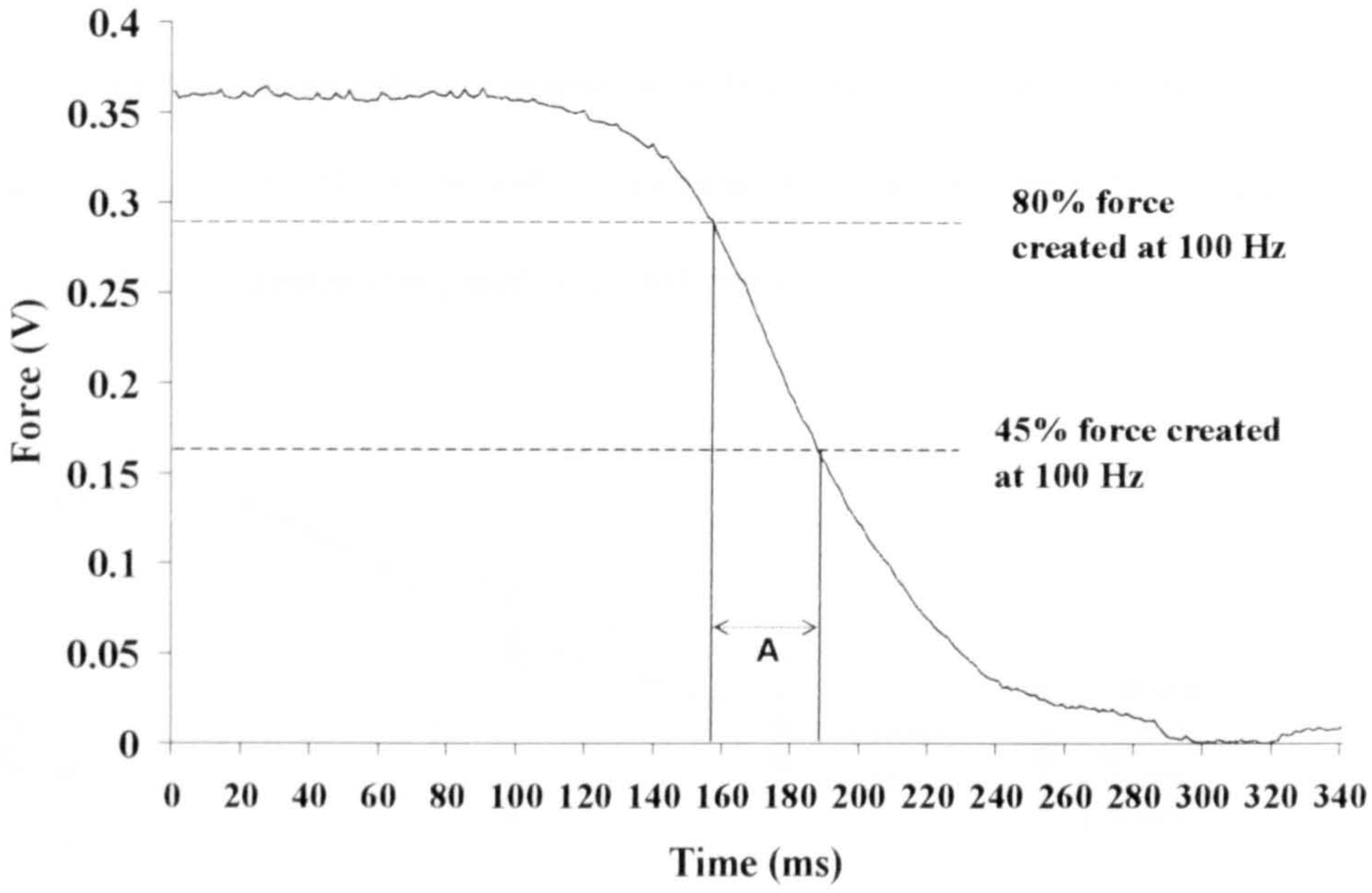

FIG. 3.13: METHOD USED TO MEASURE RELAXATION TIME FROM 80-45% FORCE CREATED AT 100 HZ (A) FROM THE RAW DATA FOR ONE SCI PERSON.

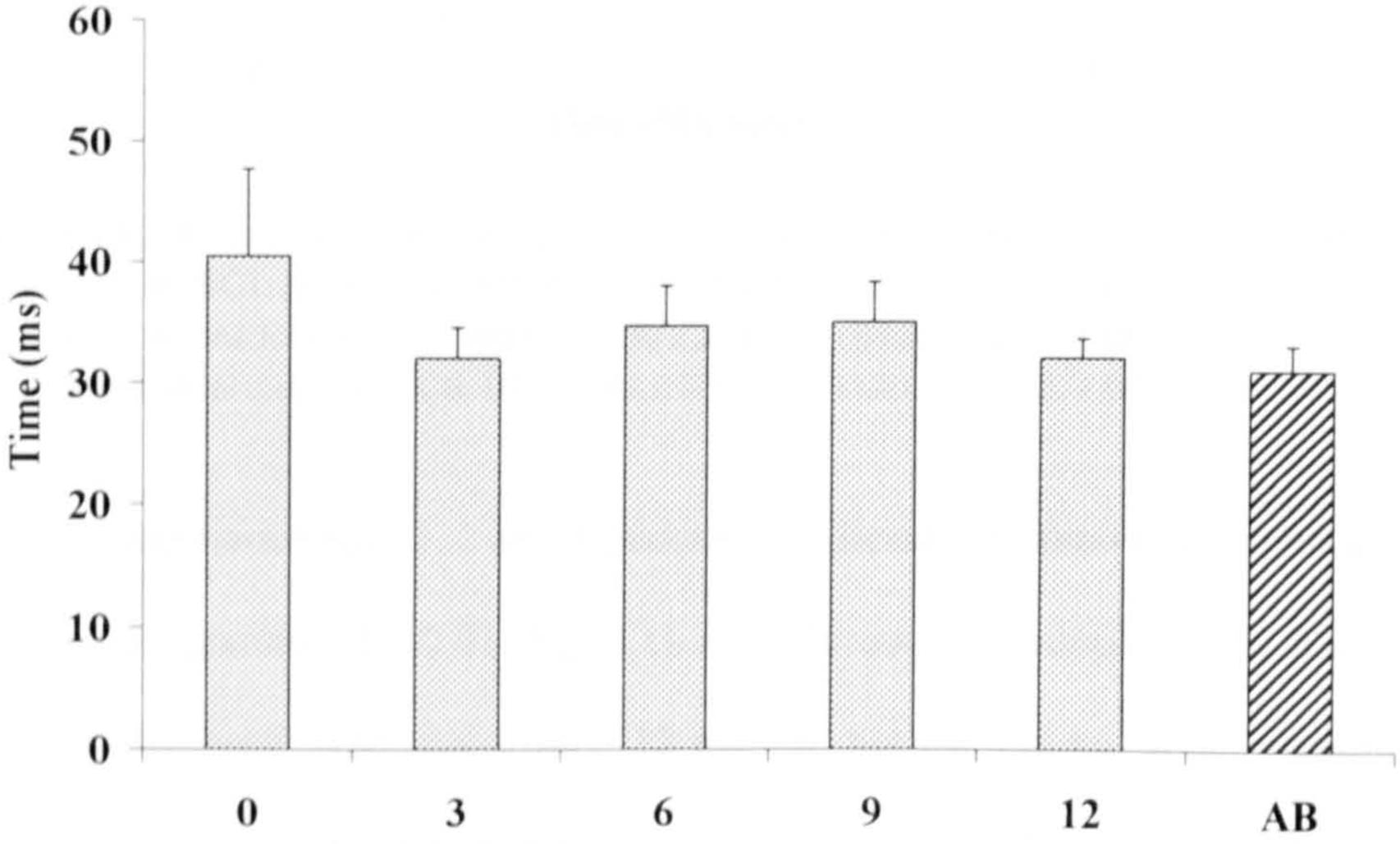

FIG. 3.14: TIME TAKEN (MEAN AND SEM) FOR RELAXATION FROM 80-45% TORQUE CREATED AT 100 HZ FOR SCI PEOPLE OVER THE YEAR OF TRAINING (GREY) AND FOR 10 UNTRAINED AB PEOPLE (STRIPED).

3.3.2.3 Fatigue resistance

The torque at 1, 2 and 3 minutes into the stimulation period increased substantially at 3 months compared with baseline and progressively improved thereafter (Fig. 3.15). At 9 and 12 months, fatigue resistance for SCI people appeared to be greater compared with AB people.

FIG 3.15: AVERAGE TORQUE AT 1, 2 AND 3 MINUTES NORMALISED TO INITIAL FORCE FOR SCI PEOPLE (DASHED LINES) AT BASELINE (CLOSED DIAMONDS), 3 (CLOSED SQUARES), 6 (CLOSED TRIANGLES), 9 (OPEN TRIANGLES) AND 12 (OPEN SQUARES) MONTHS AND FOR 10 UNTRAINED AB PEOPLE (SOLID LINE AND CIRCLES).

Torque maintained after 1, 2 and 3 minutes significantly increased throughout the training programme ($P < 0.01$, Fig. 3.16 a-c). Torque maintained after 1, 2 and 3 minutes increased gradually from 3-12 months, but this did not attain statistical significance. Fatigue resistance at 1 ($P < 0.05$), 2 and 3 ($P < 0.01$) minutes was significantly reduced for SCI people at baseline compared with AB people and was not significantly different thereafter. Fatigue resistance appeared to be

greater for SCI compared with AB people at 9 and 12 months but this did not attain statistical significance except force maintained after 1 minute was significantly greater for SCI people after 12 months training compared with AB people ($P < 0.05$).

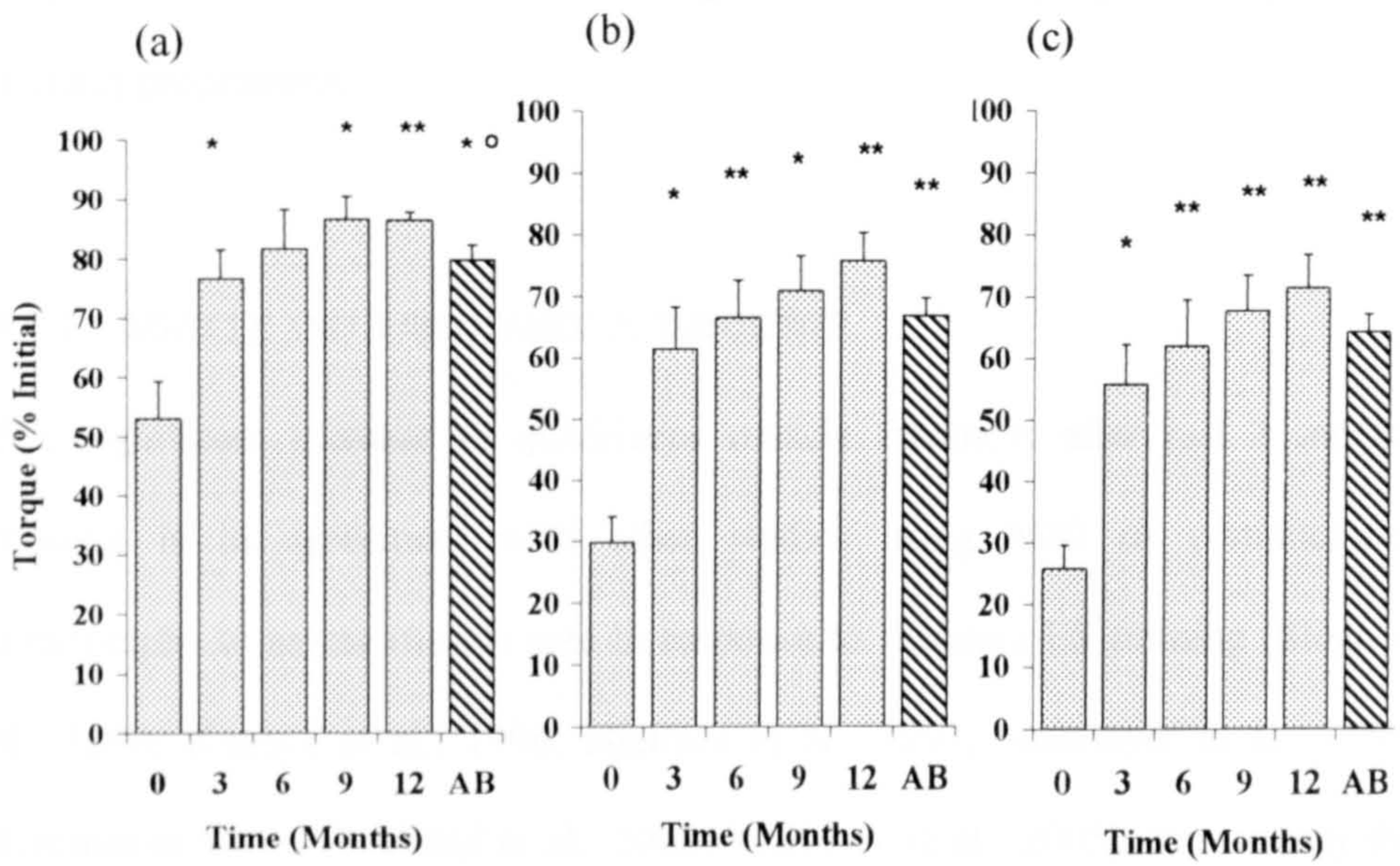

FIG 3.16: AVERAGE TORQUE AT A) 1, B) 2 AND C) 3 MINUTES NORMALISED TO INITIAL FORCE FOR SCI PEOPLE OVER THE YEAR OF TRAINING (GREY BARS) AND FOR 10 UNTRAINED AB PEOPLE (BLACK AND WHITE BAR) (SIGNIFICANTLY DIFFERENT TO BASELINE * ($P < 0.05$), ** ($P < 0.01$), SIGNIFICANTLY DIFFERENT TO 12 MONTHS ^{o} ($P < 0.05$)).

3.4 DISCUSSION

At baseline muscle size, strength and fatigue resistance were significantly lower for SCI than AB people. Subcutaneous tissue tended to be reduced but this was not statistically significant. Relative force responses at all frequencies tested were higher for SCI than AB people, but this did not attain statistical significance. After 3 months FES cycle training, muscle size, strength and fatigue resistance had improved significantly for SCI people, and continued to gradually improve thereafter. Muscle size and fatigue resistance were not significantly

different between AB and SCI people after 3 months training, but muscle strength remained significantly lower for SCI people. After 12 months, fatigue resistance appeared to be greater for SCI compared with AB people, but this was only significant at 1 minute into the test. Relative force response at all frequencies relative to 100Hz reduced gradually for SCI people throughout the training programme.

3.4.1 MUSCLE AND SUBCUTANEOUS TISSUE SIZE

The significant increase in quadriceps muscle thickness after just 3 months training is in agreement with other studies using MRI or computerised tomography to assess muscle size in response to dynamic ES training (Sloan et al., 1994; Kagaya et al., 1996; Hjeltnes et al., 1997; Neumayer et al., 1997; Scremin et al., 1999; Sköld et al., 2002; Mahoney et al., 2005). Presumably the FES cycle training placed a substantial mechanical load upon muscle groups that had previously been inactive for >2 years. It seems that this stimulated the release of growth factors and protein synthesis resulting in the observed muscle hypertrophy.

After 12 months training cross sectional area of the quadriceps and hamstrings had improved significantly. Thickness of quadriceps and calf muscles showed the greatest increase in the initial three months. Quadriceps thickness and cross sectional area had increased to a greater extent than other muscle groups after 12 months training. This was unexpected because all muscle groups were thought to be activated at the same proportion of maximum throughout the training. Presumably recumbent cycling placed a greater mechanical load on the

quadriceps muscle compared with other muscle groups measured. Alternatively it is possible that some muscle groups were not being stimulated fully or since the quadriceps are biarticular muscles, they may have been subject to eccentric activity and therefore damage. It is possible that muscle damage stimulates hypertrophy and thus might have resulted in the observed greater improvements in muscle size.

The improvement in quadriceps CSA (measured by MRI) was substantially less than the improvement in quadriceps depth (measured by ultrasound). However, when quadriceps muscle depth was measured from the MRI data, both the absolute values and relative improvements were not significantly different compared with the ultrasound data, indicating that the two methodologies are comparable. Therefore this discrepancy was probably due to the difference between a cross sectional and linear measurement, particularly given the variable changes in the areas of RF, VM, VI and VL after 12 months training (Fig. 3.6).

After 12 months FES cycle training, quadriceps muscle thickness was slightly lower for SCI compared with AB people, although this was not significant. This might be due to the long duration of training carried out (1 hour per session). This type of training is more a stimulus for endurance training adaptations than strength, which would limit improvements in muscle size in order to improve oxygen diffusion for aerobic metabolism. Alternatively, it is possible that the ES did not activate all muscle fibres during training. MRI analysis showed variable increases in individual muscles of the quadriceps for each subject (Fig 3.6), suggesting that there was not a consistent recruitment of muscle across subjects.

The inter subject variability was probably due to differences in muscle size relative to electrode size at the start of the study and difference in electrode positioning.

Subcutaneous tissue depth (ultrasound) and cross sectional area (MRI) reduced slightly as a result of training for all muscle groups measured. This is in agreement with previous studies (Scremin et al., 1999; Sköld et al., 2002) however one study did show a significant reduction in subcutaneous tissue (Hjeltnes et al., 1997). The majority of training was carried out at a respiratory exchange ratio > 1.0 (Chapter 6) indicating that carbohydrates were the primary fuel during training. This may have limited the extent of fat metabolism and thus the reduction in subcutaneous tissue. In addition, SCI people develop considerable intramuscular fat due to chronic inactivity (Fig. 3.4), therefore it is likely that some mobilisation of this intramuscular fat occurred as a result of training.

3.4.2 MAXIMAL TORQUE

At baseline, maximal torque was very low for SCI people (13.8 (2.8) Nm compared with 331.1 (27.7) Nm for AB people). However this value was similar to those previously reported for untrained SCI people measured with femoral nerve (Kagaya et al., 1996) and surface stimulation (Fornusek & Davis, 2004). However, Gerrits et al. (1999) reported significantly higher values in SCI people than those reported here (135 Nm). The reason for this discrepancy is unclear. Possibly, the more optimal knee angle used in that study (100°) gave improved

maximum torque values or the subjects in that study might have done some training prior to the study.

Maximal torque improved significantly at 3 months compared with baseline, in agreement with previous studies (Rochester et al., 1995a; Crameri et al., 2002; Gerrits et al., 2002) and continued to gradually improve thereafter. Changes through out the year correlated significantly with changes in muscle thickness. However, ES elicited maximal force in SCI people was significantly less compared with AB people at baseline and throughout the year's training. It is possible that SCI people remained significantly weaker because activation of the entire muscle group does not occur with ES during the measurement of maximal torque. Indeed, in one SCI person surface stimulation of the quadriceps elicited 73% of the torque created using femoral nerve stimulation (unpublished observations). Most AB people are able to voluntarily activate the entire muscle group during maximal voluntary contraction tests (Rutherford et al., 1986). In addition, muscles were probably not fully activated during FES cycle training for SCI people thus the mechanical stimulus for muscle growth would be limited to the activated motor units only. Also the training carried out by SCI people was perhaps more likely to increase endurance than strength.

As shown in Fig. 3.8, subject 5 experienced greater improvements in muscle strength than other subjects. Improvements in muscle strength are related to the amount of force combined with the duration of training. It has been reported that FES cycling at a lower cadence results in greater torque production. Since subject 5 cycled at a lower cadence than other subjects throughout the training

programme (see Chapter 2) it is likely that this resulted in greater relative forces being generated and the greater improvements in strength.

3.4.3 TORQUE:FREQUENCY RELATIONSHIP

Relative torque created at 1 Hz amongst SCI people at baseline was very variable (range 13-76 %). Average relative torque produced at 1 Hz for AB people was 19 % indicating that some, but not all, SCI people show an unusually large twitch response, as reported previously in paralysed muscle of SCI people (Gerrits et al., 1999). This apparent increase in activation per impulse was possibly due to i) alterations in phosphorylation of myosin light chains might have resulted in changes in cross bridge function (faster rates of attachment and development of force); ii) increased calcium release due to high proportions of fast twitch fibres; or iii) increased affinity of troponin for free calcium. The substantially larger twitch size noted in some subjects (Subjects 1, 3 and 5) did not appear to be related to age, lesion level or time since injury. It is interesting to note however that they were the weakest subjects and experienced difficulty in building cycling ability during the initial three months of training (Chapter 2). The large calcium flux in response to ES might have lead to calcium accumulation in the mitochondria in these subjects. This impairs ATP generating capacity (Kuipers, 1994) and presumably force generation during exercise.

At baseline SCI people also produced relatively more torque at 10, 20 and 50 Hz compared with AB people resulting in a leftward shift in the torque:frequency relationship, which is a function of the large twitch. However, this is an unexpected finding considering that histochemical staining has demonstrated that

chronically (>1 year) paralysed muscle is composed predominantly or exclusively of fast fibres (Grimby et al., 1976; Martin et al., 1992; Round et al., 1993; Greve et al., 1993; Rochester et al., 1995b; Chilibeck et al., 1999). Similar results have been reported previously in paralysed muscles of SCI people (Gerrits et al., 1999) but the reason for this paradox remains unclear.

Since the force:frequency relationship is affected by muscle length it is possible that SCI people experience altered optimum muscle lengths, which might result in a leftward shift of the force-frequency relationship. However it has been reported that there are no significant differences in optimum muscle length for force generation between SCI and AB people (Gerrits et al., 2005). Alternatively, Gerrits et al (2000a) suggested that the re-uptake of calcium by the SR is impaired in fast muscles of SCI people due to long periods of inactivity. This would result in increased of relaxation times and thus relatively greater torque at low frequencies.

Three out of the 5 SCI subjects had an unusually large twitch and appeared to have a greater leftward shift in the force-frequency relationship compared with other subjects. It therefore seems likely that these individuals had a greater proportion of fast muscle fibres causing the larger twitch. The leftward shift at low frequencies might be due to the size of the twitch since peak as opposed to mean force was measured at all frequencies.

With training, force relative to 100Hz generated at all frequencies reduced, resulting in a rightward shit of the force-frequency relationship indicating a slow

to fast conversion. This is unexpected considering ES training programmes have been reported to induce a fibre type conversion from IIx to IIa in both AB (Pérez et al., 2002) and SCI (Greve et al., 1993; Andersen et al., 1996; Crameri et al., 2002) people. The shift was however towards the force-frequency relationship observed in AB people, indicating that ES training altered the contractile process in SCI people to more closely match that of AB people. Previous studies have reported similar findings (Gerrits et al., 2002). This was probably due to the reduction in average twitch size with training, as has been reported previously (Gerrits et al., 2000a). Twitch size was comparable with AB people after 12 months.

Relaxation times from tetani (Fig. 3.14) also tended to reduce with training, which may have resulted in relatively lower forces at all other frequencies, shifting the force-frequency relationship rightwards. Finally, the torque-frequency relationship might have shifted rightwards (and relaxation rate from tetani reduced) due to an increase in blood flow and capillarisation after training resulting in increased muscle temperature, which causes faster rates of relaxation (Gerrits et al., 2000b).

Alternatively, changes in tendon properties as a result of SCI might affect the measured contractile properties. FES exercise might result in reduced tendon stiffness, which would affect force generation because the muscle needs to shorten further to engage the tendon and to transmit force. This results in smaller twitches and perhaps the observed rightward shift in the torque:frequency relationship.

3.4.4 FATIGUE RESISTANCE

Relative torque produced over 3 minutes was significantly greater for AB compared with SCI people at baseline. In agreement with this other studies have reported substantial reductions in fatigue resistance in SCI compared with AB people (Rochester et al., 1995a; Hillegass & Dudley, 1999; Gerrits et al., 1999; 2000), which is due to a slow to fast fibre type transformation as a result of prolonged electrical inactivity. Adaptations such as reduced blood flow, capillarisation, oxidative enzymes and mitochondria would also contribute to the low fatigue resistance observed in SCI people at baseline.

Following 3 months FES cycle training fatigue resistance improved significantly for SCI people and was not significantly different compared with AB people. This is in agreement with previous short-term ES training studies in SCI people (Stein et al., 1992; Rochester et al., 1995a; Gerrits et al., 2000a; Harridge et al., 2002). A 10-24 week FES cycle training programme has been reported to induce a fibre type conversion from IIx to IIa (Greve et al., 1993; Andersen et al., 1996; Crameri et al., 2002), with small increases in type I fibres (Martin et al, 1992; Crameri et al., 2002).

Increased blood flow to paralysed limbs of SCI people has also been found to occur rapidly (<6 weeks) following an ES training programme (Taylor et al., 1993; Gerrits et al., 2001) and capillary:fibre ratio has been reported to increase significantly following 8-10 weeks FES cycle training (Chilibeck et al., 1999; Crameri et al., 2002) but these changes have been found to be variable between subjects (Martin et al., 1992). Sabatier et al. (2006) reported no increase in blood

flow or arterial size in response to ES training in SCI people, despite improved fatigue resistance. It is possible that the low training volume (8 minutes per week) used in that study was insufficient to induce vascular adaptations. However this indicates that a mechanism other than, or in addition to, improved blood flow can bring about improvements in fatigue resistance.

Increased enzymes associated with oxidative metabolism as a result of ES training have been reported to occur by 4-24 weeks into an ES training programme (Martin et al., 1992; Rochester et al., 1995a; Kjær et al., 2001). It is therefore likely that similar adaptations occurred in the present study due to the FES cycle training resulting in the observed improvements in fatigue resistance after 3 months training.

Changes in fatigue resistance appeared to plateau after 3 months although small progressive improvements were evident. In fact SCI people appeared to be more fatigue resistant following 12 months training than AB people, but this was only significant after one minute into the fatigue test. This has not been reported previously, presumably because the duration and frequency of training used in this study was greater compared with previous studies. Stein et al. (1992) reported that increasing stimulation duration from 45 minutes to 2 hours resulted in significant increases in fatigue resistance (from 60-70 % force maintained after 3.5 minutes). This improved further when stimulation was increased to 8 hours per day with 80 % force maintained after 3.5 minutes, which is comparable to fatigue resistance reported in this study for two out of the 5 SCI subjects. It is therefore likely that the high training frequency and duration used in this study

over a one year period resulted in greater improvements in fatigue resistance than has been reported previously.

Kjær et al. (2001) reported that glycolytic (lactate dehydrogenase and hexokinaes) and oxidative (hydroxyacyl-3-dehydrogenase) enzymes improved significantly until 3 months into a one year ES training programme and did not significantly change thereafter. This indicates that improvements in these enzymes may not have caused the observed improvements in fatigue resistance after 3 months in the present study. However, the study by Kjær et al. (2001) involved a lower training frequency (3 sessions per week) than used in this study (5 sessions per week).

Substantial increases in succinate dehydrogenase (SDH) activity following a short term period (<6 months) of ES in SCI people have been reported, reaching levels that are at least comparable with AB people (Martin et al., 1992; Rochester et al., 1995a; Gerrits et al., 2003). It is unclear whether further improvements would occur after 6 months, however fatigue resistance has been found to be significantly related to the activity of SDH (Gerrits et al., 2003). Substantial increases in SDH activity as a result of training have been noted in some SCI people with an improvement of 161 % reported in one subject (Rochester et al., 1995a). Two out of the 5 subjects in the present study appeared to experience greater improvements in fatigue resistance, with 80 and 81 % initial force maintained after 3 minutes compared with 51, 70 and 73 % for the remaining subjects. AB people maintained 64 ± 2.9 % initial force after 3 minutes. It is

therefore possible that some SCI people experienced a substantially greater improvement in SDH activity as a result of the training.

The type IIx to IIa fibre type transformation that occurs after 3-6 months of ES training has been reported to occur to a greater extent after 12 months training with 44 and 91 % of fibres containing MHC type IIa only after 6 and 12 months, respectively (Andersen et al., 1996). It is therefore likely that continued fibre type transformation over the one year period resulted in the observed improvements in fatigue resistance. Furthermore, the higher training frequency and duration used in this study compared with Andersen et al. (1996) might have induced some II to I fibre type transformation after one year. Indeed, Harridge et al. (2002) reported progressive increases in the number of fibres containing the mRNA transcript for MHC I during 2-9 weeks ES exercise carried out two hours per day, 5 days per week. Fast to slow fibre type transformation is associated with improved mitochondrial density, which would substantially improve the oxidative capacity of muscle.

3.5 Conclusions

At baseline muscle size, strength and fatigue resistance was significantly reduced for SCI compared with AB people. Also SCI people produced relatively more force at 1, 10, 20 and 50 Hz compared with AB people resulting in a leftward shift in the force-frequency relationship.

The most substantial changes in muscle properties occurred after 3 months training with significant improvements in muscle size, strength and fatigue

resistance. After 3 months training, muscle size and fatigue resistance were not different compared with AB people but muscle strength remained significantly lower. This is probably due to ES failing to recruit all muscle fibres of the quadriceps before spreading to antagonistic muscle groups in SCI people. Force produced at 1, 10, 20 and 50 Hz tended to reduce with training indicating that muscle became faster, which was unexpected. This is possibly due to the reduced twitch size, faster relaxation times from tetani or due to improvements in tendon stiffness. Fatigue resistance was similar to AB people after 6 months training and appeared to be greater after 9 and 12 months, which has not been reported previously. This is probably due to continued fibre-type conversion resulting in improved mitochondrial density as well as increased SDH activity in some SCI people as a result of an intense long-term FES cycle training programme.

The following chapter reviews current literature concerning cardiopulmonary adaptations in response to FES cycling in SCI people. Cardiopulmonary adaptations as a result of the present FES cycling programme, as assessed by both incremental and constant load exercise tests, are also presented and discussed.

Chapter 4 The effects of a one year FES cycle training programme on cardiopulmonary fitness

The work described in this chapter investigated the cardiopulmonary adaptations over the one year FES cycle training programme by SCI people. A preliminary study also investigated the validity of a breath-by-breath metabolic analysis system, the Metamax 3B.

4.1 Literature Review

FES cycling provides a means for SCI people to exercise the large muscle groups of the lower limbs. Upper body exercise alone fails to elicit a substantial cardiopulmonary response and thus SCI people experience considerable reductions in cardiopulmonary fitness and an increased risk of cardiovascular diseases and diabetes. Upper body exercise also induces a considerable strain on the upper limbs increasing the risk of injury in a population already susceptible to shoulder pathology. FES cycling provides an opportunity for SCI people to stress their cardiopulmonary system without the risk of upper body injury. Furthermore, it provides an opportunity to perform a functional movement with their paralysed limbs.

Significant increases in oxygen uptake ($\dot{V}O_2$) have been noted following both short (Pollack et al., 1986; Pollack et al., 1989; Arnold et al., 1992; Goss et al., 1992; Hooker et al., 1992) and long-term (Mohr et al., 1997; Mutton et al., 1997) FES

cycling programmes, indicating improvements in cardiovascular fitness. Mohr et al. (1997) noted that no further improvements in maximum $\dot{V}O_2$ occurred after 6 months of training. In contrast, Ragnarrson et al. (1988) noted no significant change in peak $\dot{V}O_2$ following a short-term (3 month) programme of FES cycling. This is probably due to methodological differences as exercise tests in the study by Ragnarrson et al. were carried out during arm exercise. Significant increases in peak ventilation (V) have also been noted following a short term FES training programme (Pollack et al., 1989; Hooker et al., 1992; Petrofsky & Stacy, 1992). Peak respiratory exchange ratio (RER) has been reported to show a significant increase (Pollack et al., 1986; Arnold et al., 1992) as a result of FES training indicating an improved aerobic capacity. Other studies however have found no significant change in RER (Mohr et al., 1997).

Short-term FES cycle training has been reported to bring about significant increases in peak heart rate (Pollack et al., 1989, Faghri et al., 1992; Hooker et al., 1992), cardiac output (Faghri et al., 1992; Hooker et al., 1992) and reductions in submaximal heart rate at a given work load (Faghri et al., 1992; Mutton et al. 1997). It has been suggested that improved stroke volume is brought about by improved venous return due to the activation of the venous muscle pump (Faghri et al., 1992). Following a period of FES cycling, no further reductions in submaximal HR were reported after a period of hybrid (combined arm and leg) exercise by Mutton et al. (1997), providing evidence that these changes are due to adaptations in peripheral circulation as opposed to central alterations.

Petrofsky & Stacy (1992) however reported reductions in peak heart rate and no significant change in cardiac output following 6 months of FES cycling. Sloan et al. (1994) noted very small increases in HR during FES cycling in people with both complete and incomplete SCI's and attributed this to either decreased sympathetic activity or an inadequate intensity of work performed by weak muscles.

Few studies have reported changes in submaximal cardiopulmonary responses as a result of an FES training programme. Faghri et al. (1992) found no changes in resting or submaximal $\dot{V}O_2$, V or RER following a three month programme. Significantly greater SV and reduced HR (Ragnarsson et al., 1988; Faghri et al., 1992) during submaximal exercise, and lowered resting blood pressure (Faghri et al., 1992) as a consequence of training have been shown, indicating improvements in cardiovascular fitness.

ES exercise appears to be less efficient than voluntary exercise (~2-14 and 20-29 %, respectively) (Glaser et al., 1989; Goss et al., 1992; Petrofsky & Stacy, 1992; Åstrand et al., 2003). It has been reported that AB people cycling voluntarily and involuntarily (ES with epidural anaesthesia) at similar $\dot{V}O_2$ achieved significantly lower power output during involuntary exercise (~70-120 and 20-40 W during voluntary and involuntary exercise, respectively). Higher [lactate] and [H^+] (Kim et al., 1995a; Hamada et al., 2004), glycogen breakdown (Kim et al., 1995b; Kjær et al., 1996a), peripheral glucose uptake (Hamamda et al., 2004) and carbohydrate oxidation (Hamada et al., 2004) at similar $\dot{V}O_2$ rates have been noted during ES

compared with voluntary exercise. This indicates a greater contribution of anaerobic pathways for energy production. For SCI people, high relative proportions of fast twitch muscle fibres, reduced capillarisation and venous pooling in the lower limbs might further contribute to a reduced ability to work aerobically.

A period of ES training has been shown to cause significant improvements in the glycolytic and oxidative enzyme activity levels in paralysed muscles (Martin et al., 1992; Kjær et al., 2001a) and to significantly increase capillary density (Martin et al., 1992; Crameri et al., 2002). These adaptations suggest an improved ability of aerobic pathways in energy production. This is reflected in the significantly improved fatigue resistance noted following a period of FES exercise (Martin et al., 1992; Stein et al., 1992; Gerrits et al., 2000; Rochester et al., 1995a; Gerrits et al., 2002; Harridge et al., 2002). However, only small improvements in power output have been reported following a training programme (Mohr et al., 1997; Jeon et al., 2002) and the reason for this remains poorly understood.

It was hypothesised that the long-term intense (1 hour, 5 times per week) training regimen would result in significant improvements in power output and more pronounced cardiopulmonary adaptations. This chapter assessed the power output and cardiopulmonary changes at three monthly intervals throughout the one year training programme of the SCI subjects. At each time point, metabolic responses were measured during incremental and constant load exercise tests using the

portable Metamax 3B system. A preliminary study investigated the validity of the Metamax 3B in healthy controls.

4.2 VALIDITY STUDY OF METAMAX 3B

4.2.1 INTRODUCTION

An individual's $\dot{V}O_2$ is a measure of aerobic capacity. Measurements of $\dot{V}O_2$ and carbon dioxide production ($\dot{V}CO_2$) are commonly used to assess physiological efficiency during exercise or routine physical tasks. Respiratory exchange ratio (RER), the relationship between $\dot{V}O_2$ and $\dot{V}CO_2$, is a useful measure of indirect calorimetry (Wilmore et al., 1976; Bursztein et al., 1989).

The standard methodology for measuring these variables uses a direct pulmonary gas-exchange measurement system to measure the volume of air inspired or expired through a mouthpiece. Mixed expired air is collected into Douglas Bags and/or a mixing box, and samples are taken for analysis of expired oxygen and carbon dioxide. $\dot{V}O_2$ and $\dot{V}CO_2$ are then calculated using volume of inspired or expired air (VI or VĖ, respectively) and assumed values for volume of inspired O_2 and CO_2. Although this methodology has long been considered accurate and valid, it is laborious and time consuming to use and allows only relatively low sampling frequencies. The equipment is large and not mobile, limiting its use to laboratory based experiments.

A number of portable systems that provide real-time breath-by-breath analysis have now been developed. Although such systems are far more versatile, some have been shown to give invalid measurements (for review see Macfarlane, 2001). The Cosmed K2 (CosMed Corp., Rome, Italy), a portable telemetric device, has been shown to give significantly lower values of $\dot{V}O_2$ compared with a standard metabolic cart (Peel & Utsey, 1993). The system is also limited since $\dot{V}CO_2$ is not directly measured so the assumption that RER = 1 is used to calculate $\dot{V}O_2$. The Cortex X1 (Cortex Biophysik GmbH, Leipzig, Germany) has been shown to accurately determine $\dot{V}O_2$ and $\dot{V}CO_2$ during graded cycle ergometry (Schultz et al., 1997) but it was noted that the weight of the system might affect cardiorespiratory responses during weight-bearing exercise. The Metamax II (Cortex Biophysik GmbH, Leipzig, Germany) is a portable metabolic measurement system that has been shown to be valid for metabolic gas measurements up to workloads of at least 250 W (Larsson et al., 2004). Cortex Biophysik GmbH has since developed the Metamax 3B system. This portable telemetric device is lightweight and provides real-time breath-by-breath measurements including $\dot{V}O_2$, $\dot{V}CO_2$, V and RER. To my knowledge no studies validating this device have yet been published.

The aim of this preliminary study was to validate measurements of $\dot{V}O_2$, $\dot{V}CO_2$, V and RER given by the Metamax 3B against criterion values from a direct pulmonary

gas-exchange measurement system at low to moderate exercise intensities in healthy subjects.

4.2.2 METHODOLOGY

Five healthy subjects (3 female) aged 34.6 ± 2.7 (mean ± SEM) years, 171.6 (2.2) cm tall and 70.4 (2.2) kg in body mass participated. Each subject gave informed consent and carried out a health questionnaire prior to participation. No strenuous exercise was carried out 24 hours prior to testing and subjects were asked to refrain from eating for 2 hours prior to the test.

4.2.2.1 Exercise protocol

Each subject carried out a submaximal, graded exercise test on a cycle ergometer on two occasions 1-7 days apart. The tests consisted of a warm up at 25 W for 2 minutes followed by 3 stages at increasing workloads chosen to stress from 40-70 % of the subjects' maximal $\dot{V}O_2$ (Table 4.1). Each stage lasted 4 minutes in order to attain a steady state. Subjects were asked to cycle at a constant cadence of 60rpm throughout the test and were withdrawn from the test when heart rate was ≥140 bpm due to ethical committee restriction. This was sufficient for the purpose of the present study because FES exercise does not elicit HR >140 bpm in SCI people. Additionally, it was assumed that a steady state could not be achieved above this intensity (i.e. ~70 % HR_{max}). Experimental conditions were kept as consistent as possible across the 2 test sessions for each subject.

Stage	Workload (W)	
	Male	Female
1	50	50
2	100	100
3	150	125

TABLE 4.1: WORKLOAD (WATTS) CARRIED OUT BY MALE AND FEMALE SUBJECTS AT EACH STAGE OF THE TEST.

4.2.2.2 Data collection:

For both tests each subject was set up on the cycle ergometer with a 3-lead ECG (Life Pulse, HME) to measure heart rate.

Two different methods were used for respiratory data collection across the two sessions; i) the Servomex fast response O_2 and CO_2 analyser (series 1400; Servomex International Ltd, Jarvis Brook, Sussex, UK.), a Mercury flow/volume integrator (PK Morgan, UK) and mixing chamber and ii) the Metamax® 3B portable gas analysis system (Cortex Biophysik GmbH, Germany).

Servomex: This was the criterion measure. A mouthpiece was positioned at a comfortable distance in front of the subject and a nose clip was worn throughout the test. The subject inspired room air through tubing and a mouthpiece via a mercury flow/volume integrator that measured VI by integrating the measurement of flow detected by a Fleisch Flow meter connected to the subjects tubing. Expired air moved through a mixing chamber before samples of expired air were dried (Harvard Apparatus, Kent) and sampled via capillary tubes and analysed for fractions of

expired oxygen (F_EO_2) and carbon dioxide (F_ECO_2) using paramagnetic and infra-red sensors, respectively. Measurements of heart rate, F_EO_2, F_ECO_2 and V_I were recorded at the end of each minute. Air temperature, pressure and humidity were recorded from a thermometer, barometer and hygrometer, respectively. $\dot{V}O_2$ and $\dot{V}CO_2$ were then calculated using Equation 4.1 and converted from $\dot{V}O_2$ and $\dot{V}CO_2$ at ambient temperature and pressure in saturated air (ATPS) to standard temperature and pressure in dry air (STPD) using Equation 4.2.

Equation 4.1: Calculation of $\dot{V}O_2$ and $\dot{V}CO_2$ from the Servomex recordings.

$$\dot{V}O_2 \ (ATPS) = V_I [F_IO_2 - F_EO_2] \times [F_IN_2/F_EN_2]$$

$$\dot{V}CO_2 \ (ATPS) = V_I [F_ECO_2 - F_ICO_2] \times [F_IN_2/F_EN_2]$$

Where F_IO_2 = fraction of inspired oxygen (0.209), F_IN_2 = fraction of inspired nitrogen (0.79), F_EN_2 = fraction of expired nitrogen = $1 - (F_EO_2 + F_ECO_2)$.

EQUATION 4.2: CONVERSION OF $\dot{V}\ O_2$ AND $\dot{V}\ CO_2$ ATPS TO STPD FROM THE SERVOMEX RECORDINGS.

$$\dot{V}O_2 \text{ or } \dot{V}CO_2 \times [(P_B - P_{H2O})/760] \times [273/(273 + \dot{T}r)]$$

Where P_B is barometric pressure, P_{H2O} is vapour pressure (room temperature x room humidity) and Tr is room temperature.

O_2 and CO_2 sensors were calibrated using ambient air and 2 calibration gases, the first containing 100 % N_2 and the second with known O_2 and CO_2 concentrations of

13.69 and 5.03 %, respectively. Volume was calibrated by taking 20 measurements using 1 and 3 litre syringes.

Metamax 3B: A facemask was tightly positioned over the subjects' nose and mouth. The Metamax instrument was placed in a harness on the shoulders of the subject (Fig. 4.1). Expired air was sampled via a capillary tube and immediately analysed by the instrument, which contains electrochemical and infrared sensors for O_2 and CO_2, a barometer and a thermometer. The volume of inspired and expired air was measured by means of a digital turbine volume transducer attached to the facemask.

FIG. 4.1: METAMAX 3B AND MASK ATTACHED TO A SUBJECT.

The system is said to provide real time breath-by-breath measurements of F_IO_2, F_EO_2, F_ICO_2, F_ECO_2, VI and VĖ. Ambient temperature and pressure, expiratory temperature (Tb), environmental pressure (Pa) and P_{H2O} were also recorded

automatically. Values for $\dot{V}O_2$ and $\dot{V}CO_2$ were then automatically calculated by MetaSoft software (Cortex Biophysik GmbH, Germany) using Equation 4.3 and converted from ambient temperature and pressure in saturated air (ATPS) to standard temperature and pressure in dry air using Equation 4.4.

EQUATION 4.3: CALCULATION OF $\dot{V}O_2$ AND $\dot{V}CO_2$ BY THE METAMAX.

$$\dot{V}O_2 = (F_IO_2 \times VI - F_EO_2 \times VE) / 100$$

$$\dot{V}CO_2 = (F_ECO_2 \times VI - F_ICO_2 \times VE) / 100$$

EQUATION 4.4: CONVERSION OF $\dot{V}O_2$ AND $\dot{V}CO_2$ ATPS TO STPD BY THE METAMAX.

$$273 / Tb \times (P_{H2O} / 101.3)$$

The system was calibrated following the manufacturer's guidelines. O_2 and CO_2 sensors were calibrated using ambient air and a calibration gas with known O_2 and CO_2 concentrations of 17 % and 5 %, respectively. In addition, volume was calibrated by taking 20 measurements using 1 and 3 litre syringes.

4.2.2.3 Data Treatment:

Servomex: V was taken from the final minute at each stage. FEO_2 and $FECO_2$ measurements were taken with a pre-calculated one minute delay time to compensate for the lag caused by the mixing chamber.

Metamax: Raw breath-by-breath values for $\dot{V}O_2$, $\dot{V}CO_2$, V and RER were filtered to remove any data points >2 SD of the mean. Data was re-averaged after each individual data point was removed (Saunders, 2004; unpublished). This was in order to remove miss-triggered breaths due to interruptions in breathing patterns and caused by factors not related to exercise. Data collected during the final minute of each stage were then averaged.

4.2.2.4 Data Analysis

Paired two tailed Student t-tests were carried out on $\dot{V}O_2$, $\dot{V}CO_2$, V and RER to identify any systematic differences between calculated values from the Servomax and Metamax. A paired two tailed t-test was also carried out on heart rate data to determine any differences in work intensity across the two tests (significance level set at 0.05). Mean, standard deviation and differences as a percentage of the mean difference were calculated for $\dot{V}O_2$, $\dot{V}CO_2$, V and RER. These variables were analysed for agreement using Bland-Altman's Limits of Agreement (Equation 4.5).

EQUATION 4.5: BLAND-ALTMAN'S LIMITS OF AGREEMENT.

$$95\ \%\ \text{Limits} = (1.96 \times \text{SD of difference}) +/- \text{Mean difference}$$

Where: SD of difference = standard deviation of the difference between Servomax and Metamax; and

Mean difference = mean difference between Servomax and Metamax.

4.2.3 RESULTS

Two of the 5 subjects were unable to complete stage 3 of the test since HR $\geq$ 140 bpm. Therefore n=3 for stage three data and n=5 for all other data. There were no significant differences in heart rate across the two testing conditions (mean 107.0 and 103. 4 bpm at stage 1, 130.6 and 125.8 bpm at stage 2 and 138.7 and 130.0 bpm at stage 3 for the Servomex and Metamax, respectively, P = 0.51). Volume calibration using known volumes of 1 and 3 litres gave average values for the Servomex of 0.92 and 2.89 L and 1.04 and 3.01 L for the Metamax, respectively. Consequently, all volume data as measured by the Servomex was corrected by a factor of 0.94.

Values for $\dot{V}O_2$, $\dot{V}CO_2$, V and RER as measured by the Servomex and the Metamax 3B are shown in Table 4.2.

	Stage	Servomex Mean	SD	Metamax 3B Mean	SD	Difference (% of mean)
$\dot{V}O_2$ ($l.min^{-1}$)	Rest	0.25	0.06	0.28	0.21	0.03 (11.3)
	Stage 1	0.89	0.04	0.97	0.39	0.08 (8.6)
	Stage 2	1.30	0.20	1.34	0.35	0.04 (3.0)
	Stage 3	1.84	0.62	1.87	0.70	0.03 (1.6)
$\dot{V}CO_2$ ($l.min^{-1}$)	Rest	0.21	0.06	0.22	0.16	0.01 (4.7)
	Stage 1	0.75	0.06	0.8	0.35	0.05 (6.5)
	Stage 2	1.15	0.14	1.17	0.31	0.02 (1.7)
	Stage 3	1.73	0.64	1.73	0.64	0.00 (0.0)
V ($l.min^{-1}$)	Rest	9.5	1.81	7.8	3.2	1.7 (19.7)
	Stage 1	24.3	1.10	22.0	7.0	2.3 (9.8)
	Stage 2	32.6	2.64	32.3	9.2	0.3 (1.0)
	Stage 3	44.2	13.06	50.6	10.2	6.4 (13.5)
RER	Rest	0.86	0.05	0.8	0.12	0.06 (7.2)
	Stage 1	0.84	0.07	0.82	0.06	0.02 (2.4)
	Stage 2	0.88	0.07	0.88	0.06	0.00 (0.0)
	Stage 3	0.93	0.06	0.93	0.08	0.00 (0.0)

TABLE 4.2: OXYGEN UPTAKE ($\dot{V}O_2$), CARBON DIOXIDE PRODUCTION ($\dot{V}CO_2$), VOLUME (V) INSPIRED (SERVMEX) OR EXPIRED (METAMAX) AND RESPIRATORY EXCHANGE RATIO (RER) AS MEASURED BY THE SERVOMEX AND METAMAX 3B.

Overall differences calculated as a percentage of the mean difference for $\dot{V}O_2$, $\dot{V}CO_2$, V and RER were 4.5, 2.1, 1.9 and 2.3%, respectively. Paired t-tests revealed no significant differences between the Servomax and Metamax 3B for all variables ($P > 0.05$). Fig. 4.2 shows Bland-Altman limits of agreement plots for $\dot{V}O_2$, $\dot{V}CO_2$, V and RER.

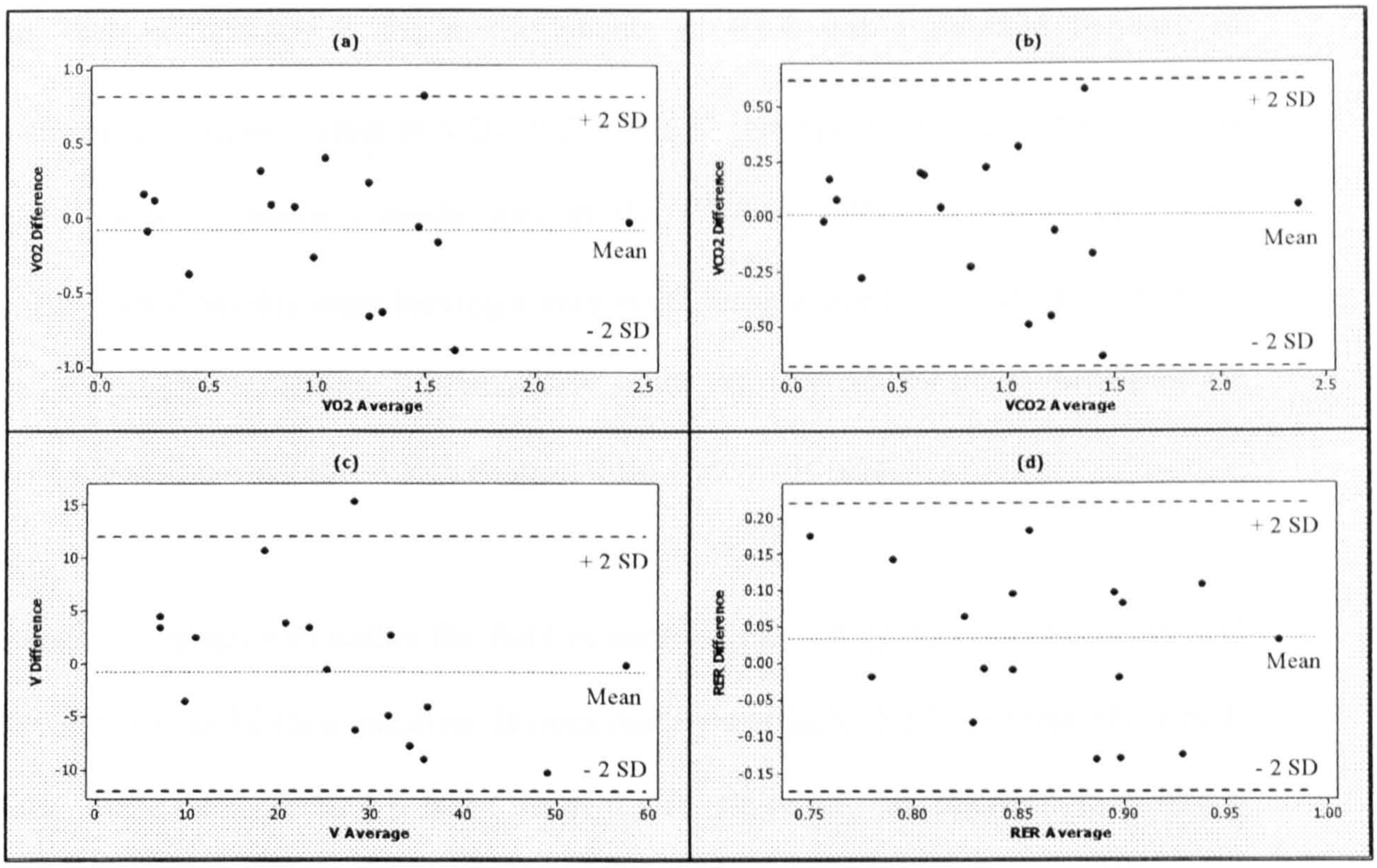

FIG. 4.2: BLAND-ALTMAN PLOTS FOR (A) OXYGEN UPTAKE, (B) CARBON DIOXIDE PRODUCTION, (C) VOLUME (V) AND (D) RESPIRATORY EXCHANGE RATIO (RER). THE DOTTED LINE SHOWS THE MEAN OF DIFFERENCES BETWEEN SERVOMAX AND METAMAX AND THE DASHED LINE SHOWS ± 2 STANDARD DEVIATIONS.

4.2.4 DISCUSSION

This study found the Metamax 3B to be a valid measurement device for $\dot{V}O_2$, $\dot{V}CO_2$, V and RER at low to moderate workloads. For all variables measured, the mean differences were small and equated to 2-5 % of the mean overall values for each variable.

Bland-Altman Limits of Agreement plots (Fig. 4.2) identify the mean difference and a 95% range calculated from the standard deviation (SD) for $\dot{V}O_2$ (a), $\dot{V}CO_2$ (b), V (c) and RER (d). Visual analysis of the plots indicates no systematic bias between

the two methodologies for measurements of RER but a possible increase in variability at higher values of $\dot{V}O_2$, $\dot{V}CO_2$ and V. However, since 2 of the 5 subjects were unable to attain a steady state at the 3rd stage (HR ≥140 bpm), they were withdrawn from this stage leaving a very small sample size (n=3), which could have caused greater variability. The relatively small values given for mean difference for all 4 parameters in Table 3 also suggest little systematic bias.

Recent developments within the field of exercise physiology have made mobile and portable O_2 and CO_2 measuring devices readily available. Such systems allow real-time measurements to be taken in real life situations, as opposed to artificial laboratory conditions. This means that the findings of scientific research can be more applicable to real life. However, it has been shown that some systems can provide invalid measurements due to limitations in the design (for review see MacFarlane, 2001). The systems are easy to use for those with little knowledge of exercise physiology and thus measurements might be taken for granted without complete understanding the validity of underpinning calculations. It is therefore important to identify how measurements are being taken and calculated by these systems and to compare these to standard pulmonary gas-exchange measurement systems.

In order to make a direct comparison between the two systems it would be ideal to measure them in series. However connection of the systems in this way may affect the accuracy of the readings from one or both of them due to the additional dead

space caused by the extra breathing valve (Larsson et al., 2003). Therefore the design chosen for this study involved two identical trials at three low to moderate exercise intensities. Although conditions were kept as controlled as possible, within subject (day-to-day) variation is still present. Since there was no significant difference between heart rates recorded across the 2 trials, it can be assumed that exercise stress levels were similar across the 2 trials. Becque et al. (1993) identified within subject variations of 4.3% and 6.8% for $\dot{V}O_2$ and V, respectively.

It should be noted that this validation study was only completed at low-moderate exercise intensities (40-70 % of the subjects' maximal $\dot{V}O_2$). Further work is required to assess the validity of the Metamax 3B at higher workloads.

In conclusion, the VO_2, VCO_2, V and RER as measured by the Metamax 3B were found to be valid compared to a direct pulmonary gas-exchange measurement system at low-moderate exercise intensities. Further work is required to assess validity at higher work intensities. On the basis of these results the Metamax 3B was used for all subsequent metabolic measurements.

4.3 METHODOLOGY

Incremental and constant load exercise tests were carried out at three monthly intervals throughout the one year training programme in order to assess changes in cardiopulmonary fitness. Peak power, peak and steady state $\dot{V}O_2$, V, RER and HR, anaerobic threshold and cycling efficiency were measured at each time point. Baseline Measurements were carried out at a different time point for exercise tests because SCI people were unable to cycle until after their initial training period. Therefore baseline data is referred to as 'post initial training' (PIT) for these data sets.

4.3.1 INCREMENTAL AND CONSTANT LOAD EXERCISE TESTS

Exercise tests were carried out by use of a feedback control system integrating electric motor assistance and functional electrical stimulation for cycling (Hunt et al., 2003). The system provides conditions of well-controlled cadence and work rate suitable for accurate cardiopulmonary testing by feedback control of cycling cadence and leg power output. The subject's power output is calculated from the force applied to the crankshaft and the cadence. This is maintained at a reference power output (according to the exercise test protocol) by adjusting the intensity (pulse width) of stimulation from motor (0 %) to maximum (100 %) threshold for each subject. Motor threshold was defined as the stimulation intensity at which the initial motor response occurred and maximal threshold was the stimulation intensity at which no additional muscle response occurred when stimulation intensity was further increased. As fatigue reduces power output the controller increases the

stimulation intensity to compensate. If cadence starts to exceed the reference cadence (50 rpm) the controller reduces the motor contribution (Fig. 4.3) to maintain the cadence. The combined power from the motor and the test subject remains constant at a set cadence. Therefore, as the contribution from the test subject changes (due to a changing power reference), constant cadence is maintained by altering the contribution from the motor (Fig. 4.4).

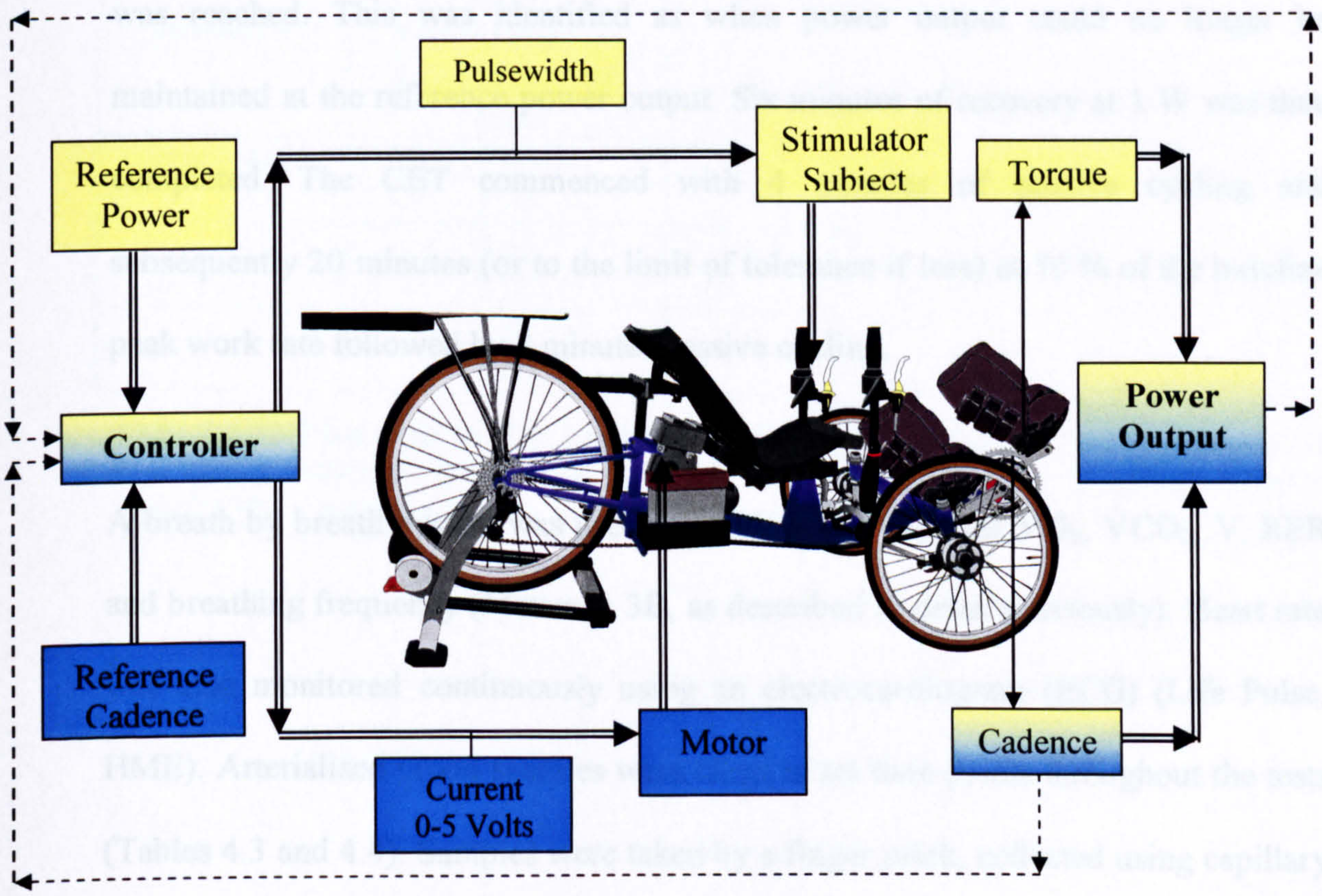

FIG. 4.3: DIAGRAM OF POWER OUTPUT AND CADENCE CONTROL FEEDBACK LOOPS FOR MOTORISED TRIKE (YELLOW = POWER CONTROL LOOP, BLUE = MOTOR CONTROL LOOP, DOUBLE ARROW = INPUT, SINGLE ARROW = OUTPUT, DASHED ARROW = FEEDBACK).

Two cardiopulmonary exercise protocols were completed at each test, an incremental exercise test (IET) and a submaximal constant load exercise test (CET).

The IET was always completed first and the CET was carried out 1-7 days later. Subjects were asked to fully comply with the training programme for one week prior to tests and not to carry out a training session on the day prior to testing. Subjects were also requested to abstain from alcohol and caffeine 24 and 4 hours before testing, respectively, and to consume a light meal at least two hours prior to tests.

The IET commenced with 4 minutes of passive cycling and thereafter work-rate was increased by 1-2 $W.min^{-1}$ every minute until saturation (100 % stimulation intensity) was reached. This was identified as when power output could no longer be maintained at the reference power output. Six minutes of recovery at 1 W was then completed. The CET commenced with 4 minutes of passive cycling and subsequently 20 minutes (or to the limit of tolerance if less) at 70 % of the baseline peak work rate followed by 8 minutes passive cycling.

A breath by breath system was used to continuously monitor $\dot{V}O_2$, $\dot{V}CO_2$, V, RER and breathing frequency (Metamax 3B, as described in detail previously). Heart rate was also monitored continuously using an electrocardiogram (ECG) (Life Pulse, HME). Arterialised blood samples were taken at set time points throughout the tests (Tables 4.3 and 4.4). Samples were taken by a finger prick, collected using capillary tubes and mixed with haemolysing solution. Samples were then analysed for blood lactate using the Super GL easy (Diasys Diagnostic Systems GmbH, Germany), which uses a biosensor to measure lactate and was calibrated according to the manufacturer's guidelines. Subjective measurements of ratings of perceived exertion

(Borg, 1970) and perceived breathlessness (Borg, 1982) were also obtained at set time points throughout the tests (Tables 4.3 and 4.4).

Phase	Time Point (Minutes)	Measurement			
		HR	BL	RPE	RPE
Rest	1.15 before E_r	x		x	x
	1.00 before E_r		x		
Passive	1.15 before E_p	x		x	x
	1.00 before E_p		x		
Ramp	0.30	x		x	x
	0.45		x		
	3.30	x		x	x
	3.45		x		
Continued every 3 minutes until...					
	0.15 before peak	x		x	x
	Peak		x		
Recovery	0.45	x		x	x
	1.00		x		
	2.45	x		x	x
	3.00		x		
	4.45	x		x	x
	5.00		x		

TABLE 4.3: TIME POINTS FOR HEART RATE (HR), BLOOD LACTATE (BL), RATES OF PERCEIVED EXERTION (RPE) AND BREATHLESSNESS (RPB) MEASUREMENTS DURING THE IET (E_R = END OF REST, E_P = END OF PASSIVE).

Phase	Time Point (Minutes)	Measurement			
		HR	BL	RPE	RPB
Rest	1.15 before E_r	x		x	x
	1.00 before E_r		x		
Passive	1.15 before E_p	x		x	x
	1.00 before E_p		x		
Constant load	9.45	x		x	x
	10.00		x		
	14.45	x		x	x
	15.00		x		
	19.45	x		x	x
	20.00		x		
Recovery	0.45	x		x	x
	1.00		x		
	2.45	x		x	x
	3.00		x		
	4.45	x		x	x
	5.00		x		

TABLE 4.4: TIME POINTS FOR HEART RATE (HR), BLOOD LACTATE (BL), RATES OF PERCEIVED EXERTION (RPE) AND BREATHLESSNESS (RPB) MEASUREMENTS DURING THE CET (E_R = END OF REST, E_P = END OF PASSIVE).

4.3.2 DATA ANALYSIS

Raw breath-by-breath values for $\dot{V}O_2$, $\dot{V}CO_2$, V and RER were filtered to remove data points >2 SD of the mean. Data were re-averaged after each individual data point was removed (Saunders, 2004; unpublished). This was in order to remove miss-triggered breaths due to interruptions in breathing patterns and caused by factors not related to exercise. 1-3 minutes of steady state rest and passive data were averaged for each test. For the IET, data collected during the final minute of the ramp were averaged. Peak values for HR, RER, BL, RPE, RPB were taken as the maximum value recorded throughout the entire test. Anaerobic threshold was assessed by plotting $\dot{V}O_2$ against $\dot{V}CO_2$ and visually assessing the point at which $\dot{V}$

CO_2 begins to increase at a greater rate than $\dot{V}O_2$. The $\dot{V}O_2$ value at the anaerobic threshold was then taken.

For the CET, minute-by-minute data were averaged for the 20-minute constant load and 8-minute recovery. Data for the final 10 minutes of the constant load were averaged to obtain steady state values. HR, RER, BL, RPE and RPB were also averaged over the final 10 minutes of the constant load. Work efficiency was calculated as shown in Equation 4.6.

EQUATION 4.6: CALCULATION OF WORK EFFICIENCY.

$$\text{Power output} / ((\dot{V}O_{2\ \text{steady state}} - \dot{V}O_{2\ \text{passive}}) \times \text{energy consumed (joules.sec}^{-1})) \times 100$$

Where; Energy consumed = $\text{Kcal.L}(O_2)^{-1}.\text{min}^{-1}$ (based on RER) x 4.185 x 1000 / 60

Analysis of variance was carried out on all $\dot{V}O_2$, V, RER, HR, BL, RPE and RPB data for both the IET and CET at each time point. Post-hoc analysis was carried out using paired Students T-Tests. Pearsons correlations were carried out to compare changes in $\dot{V}O_2$ and V with power output and training data (Chapter 2).

4.4 RESULTS

4.4.1 INCREMENTAL EXERCISE TEST (IET)

Fig. 4.4 shows a typical plot of stimulation intensity (%), reference and actual power output (W) and cadence (rpm) from an IET carried out on one subject.

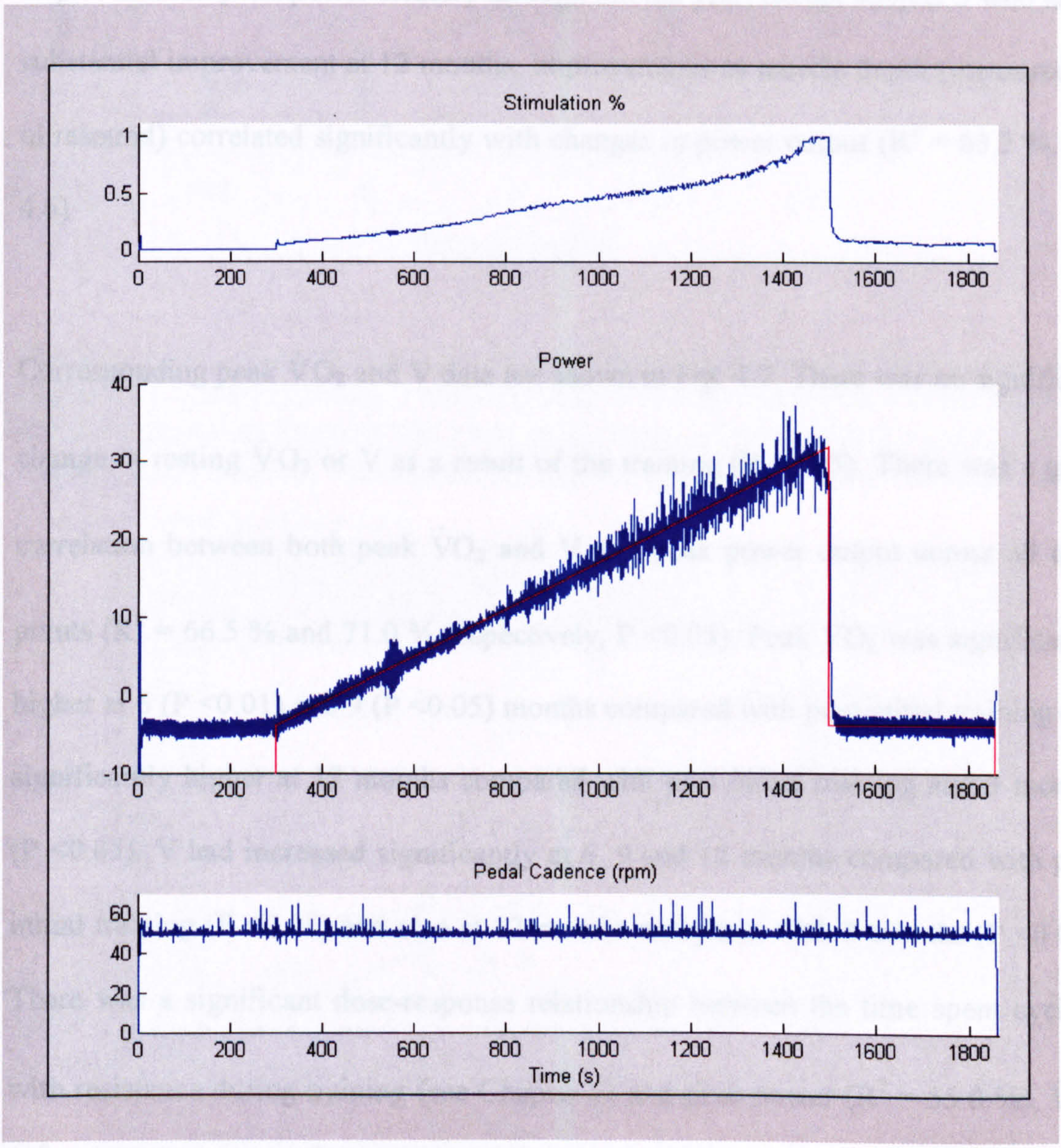

FIG. 4.4: TYPICAL PLOT OF STIMULATION INTENSITY (0-1 = 0-100%), REFERENCE (RED) AND ACTUAL (BLUE) POWER OUTPUT (WATTS) AND CADENCE FROM ONE SUBJECT DURING AN INCREMENTAL EXERCISE TEST.

Average peak power output increased steadily throughout the one year training programme (Fig. 4.5). Peak PO at 6, 9 and 12 months was significantly greater compared with 0 months ($P < 0.05$), and at 9 and 12 months peak PO was also significantly greater than at 3 months ($P < 0.05$). Subjects 2, 3 and 4 reached a plateau or peak power began to decline at 9 or 12 months. Subjects 1 and 5 improved their peak power steadily through out the year, except subject 5 who had a substantial improvement at 12 months. Improvements in muscle depth (measured by ultrasound) correlated significantly with changes in power output ($R^2 = 63.2$ %, Fig 4.6).

Corresponding peak $\dot{V}O_2$ and V data are shown in Fig. 4.7. There was no significant change in resting $\dot{V}O_2$ or V as a result of the training ($P > 0.05$). There was a good correlation between both peak $\dot{V}O_2$ and V and peak power output across all time points ($R^2 = 66.5$ % and 71.0 % respectively, $P < 0.05$). Peak $\dot{V}O_2$ was significantly higher at 6 ($P < 0.01$) and 9 ($P < 0.05$) months compared with post initial training and significantly higher at 12 months compared with post initial training and 3 months ($P < 0.05$). V had increased significantly at 6, 9 and 12 months compared with post initial training ($P < 0.01$) and also at 12 months compared with 3 months ($P < 0.05$). There was a significant dose-response relationship between the time spent cycling with resistance during training (see Chapter 2) and peak power ($R^2 = 55.0$ %), $\dot{V}O_2$ ($R^2 = 50.7$ %) and V ($R^2 = 41.9$ %) ($P < 0.05$).

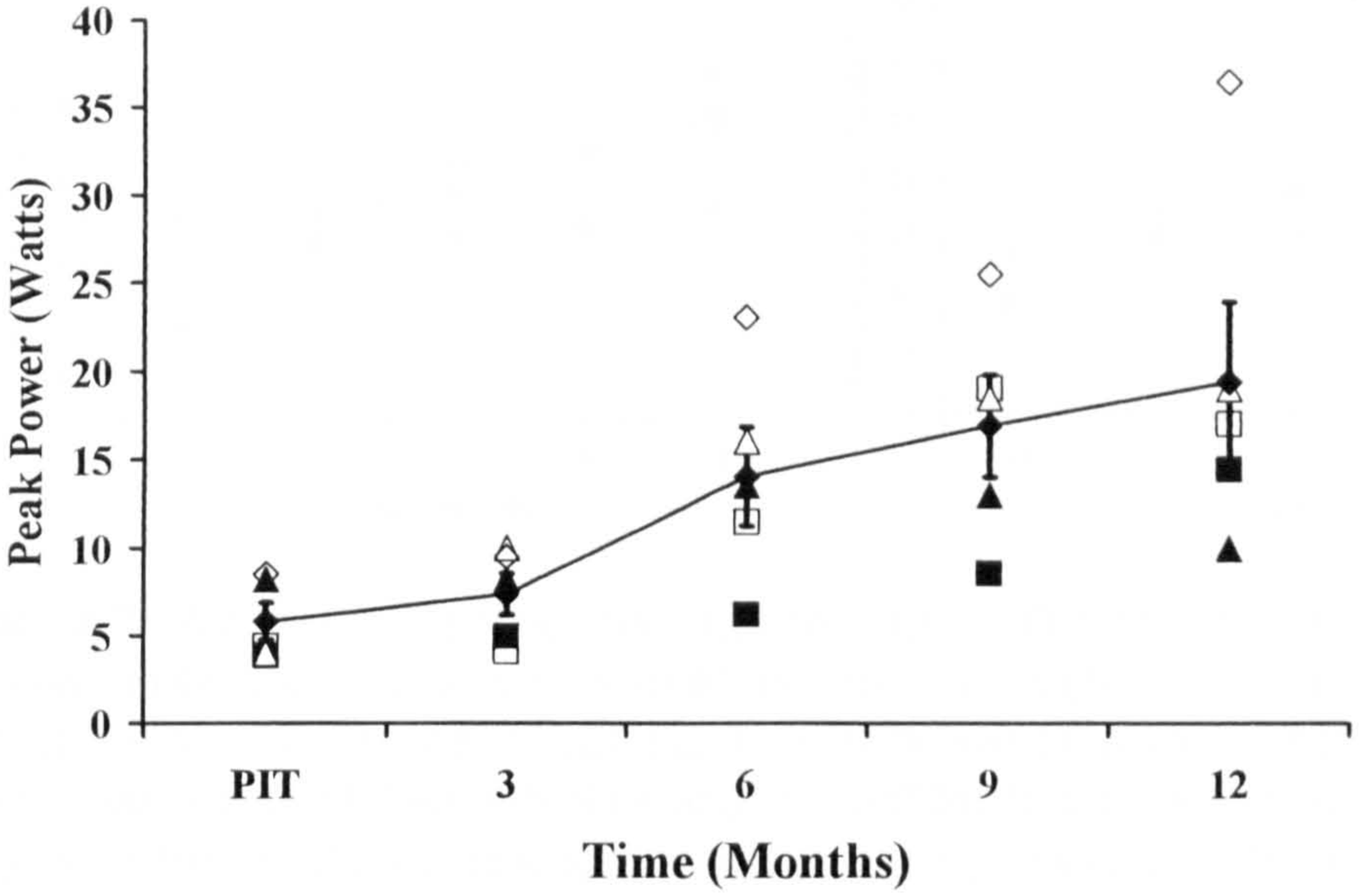

FIG. 4.5: AVERAGE PEAK POWER (CLOSED DIAMOND AND LINE) AND INDIVIDUAL DATA (SUBJECT 1 CLOSED SQUARE, 2 OPEN SQUARE, 3 CLOSED TRIANGLE, 4 OPEN TRIANGLE AND 5 OPEN DIAMOND) OVER THE YEAR OF TRAINING. (* SIGNIFICANTLY DIFFERENT TO POST INITIAL TRAINING, $^{+}$ SIGNIFICANTLY DIFFERENT TO 3 MONTHS ($P < 0.05$)).

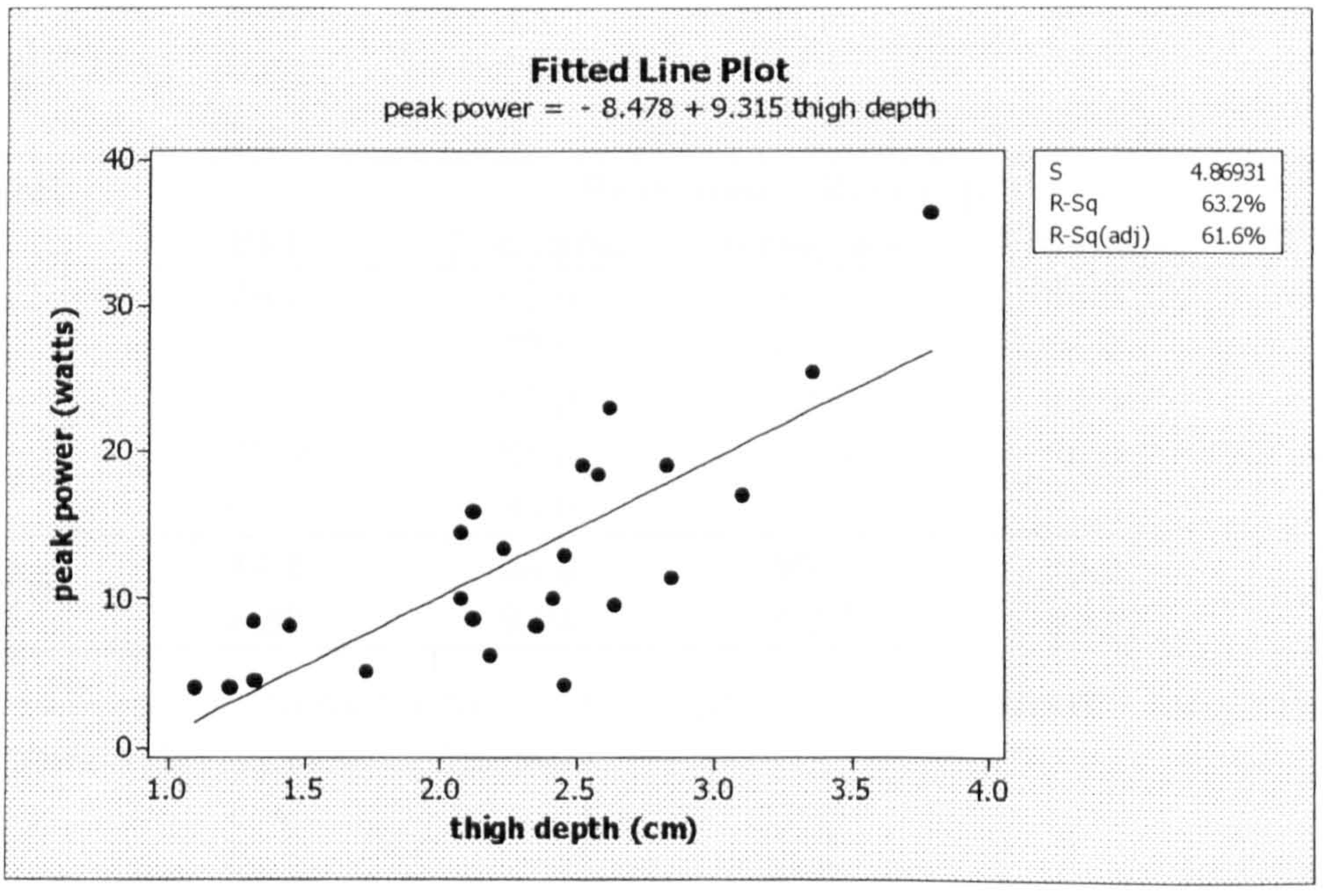

FIG. 4.6: PEAK POWER AND MUSCLE DEPTH FOR 5 SCI PEOPLE THROUGH OUT THE TRAINING PROGRAMME.

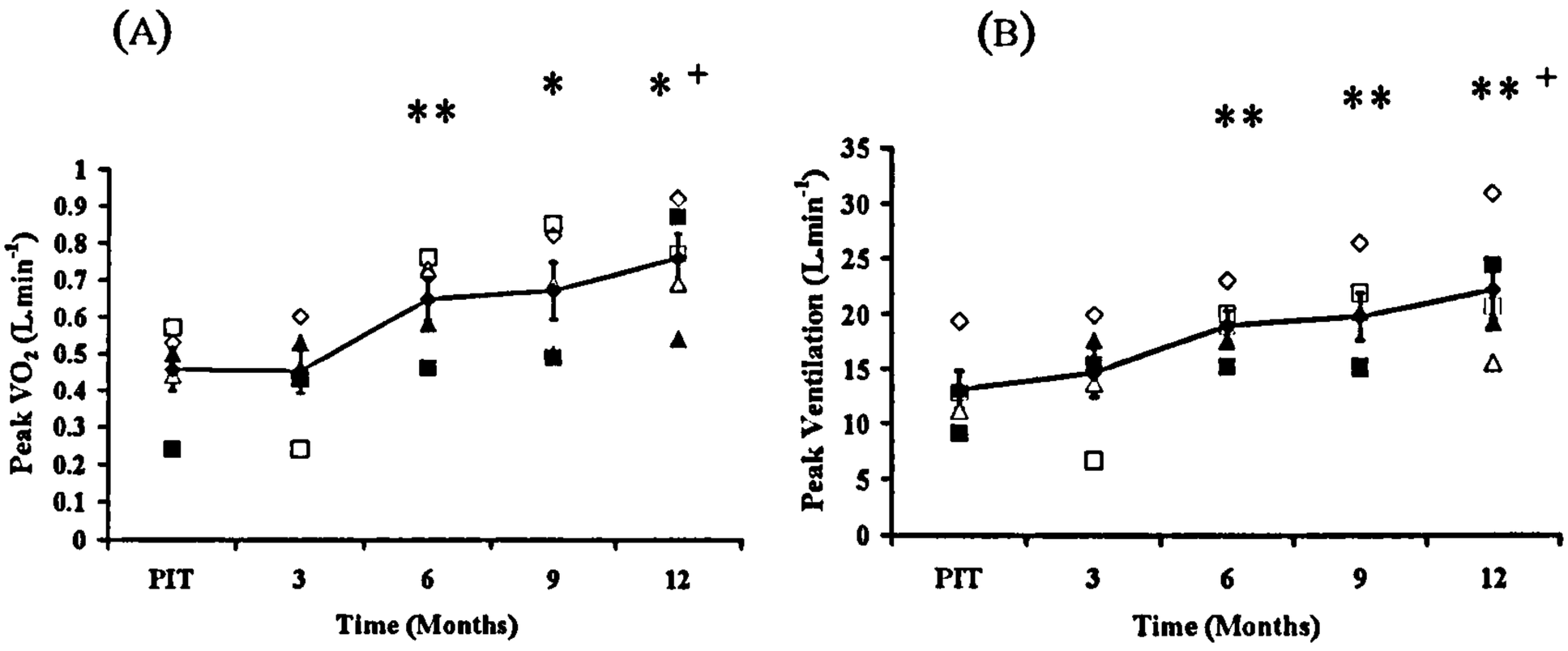

FIG. 4.7: AVERAGE (A) PEAK OXYGEN UPTAKE ($\dot{V}O_2$) AND (B) PEAK VENTILATION (CLOSED DIAMOND AND LINE) AND INDIVIDUAL DATA (SUBJECT 1 CLOSED SQUARE, 2 OPEN SQUARE, 3 CLOSED TRIANGLE, 4 OPEN TRIANGLE AND 5 OPEN DIAMOND) OVER THE YEAR OF TRAINING. (SIGNIFICANTLY DIFFERENT TO POST INITIAL TRAINING *($P<0.05$), **($P<0.01$), SIGNIFICANTLY DIFFERENT TO 3 MONTHS $^{+}$($P<0.05$)).

There was no significant change in resting or peak HR as a result of the training ($P>0.05$). There was however a trend for peak heart rate to increase during the initial 6 months of training (Table 4.5).

Subject	Peak Heart Rate (bpm)				
	PIT	3 months	6 months	9 months	12 months
1	76.0	91.0	85.0	84.0	90.0
2	--	72.0	85.0	83.0	76.0
3	81.0	93.0	90.0	89.0	83.0
4	77.0	83.0	91.0	96.0	89.0
5	90.0	93.0	95.0	98.0	108.0
Average	**81.0**	**86.4**	**89.2**	**90.0**	**89.2**
SD	**6.38**	**9.04**	**4.27**	**6.82**	**5.32**

TABLE 4.5: PEAK HEART RATE FOR SUBJECTS 1-5, AVERAGE AND STANDARD DEVIATION OVER THE YEAR OF TRAINING.

Fig. 4.8 shows raw VO_2 data plotted against VCO_2 data for one SCI subject during an IET, indicating the methodology used to assess the anaerobic threshold.

Anaerobic threshold, peak RER and lactate showed no significant change but there was a trend for them to increase throughout the training programme (Table 4.6). As can be seen in Table 4.7, peak ratings of perceived exertion and breathlessness also did not change throughout the training period for all 5 subjects.

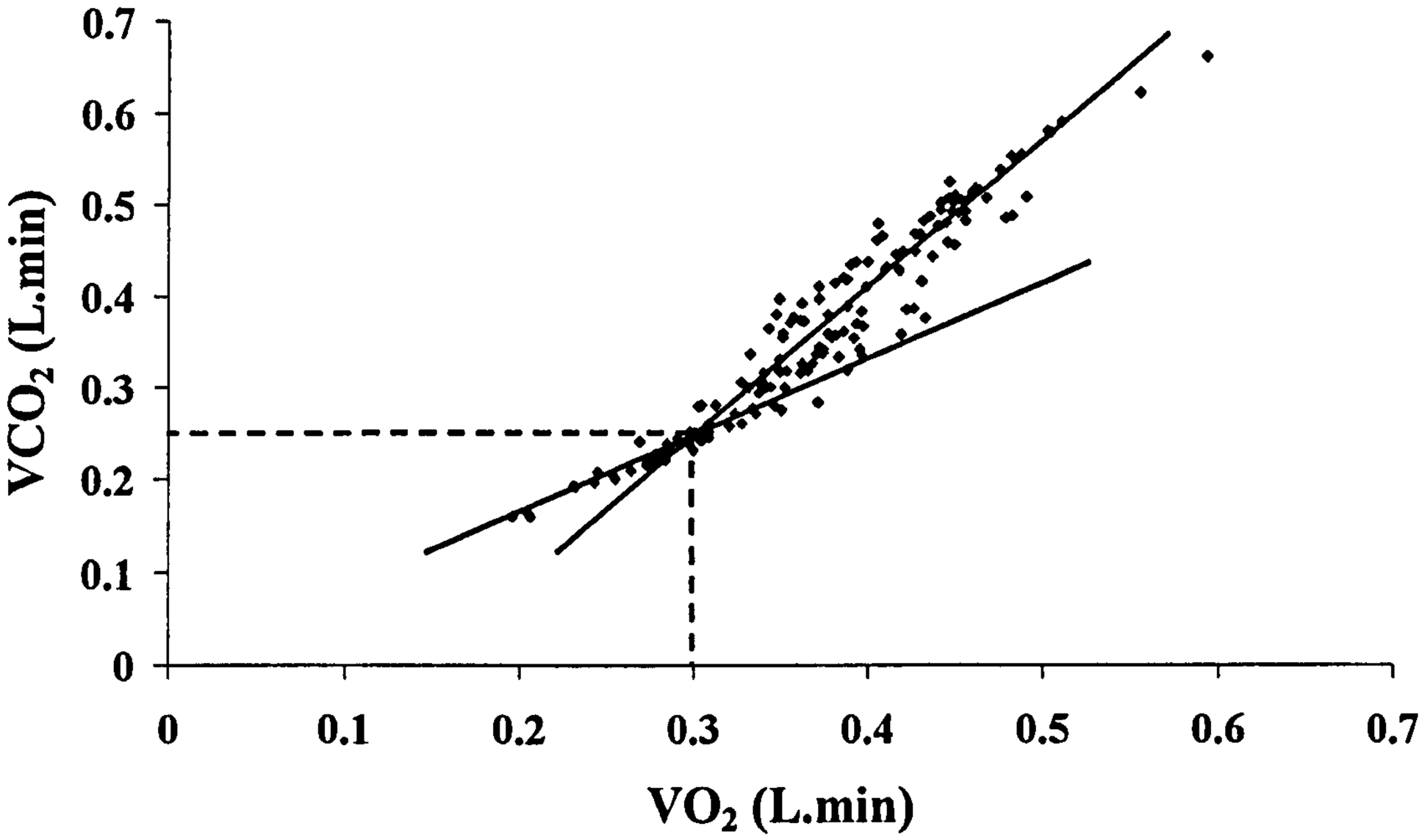

FIG. 4.8: RAW VO_2 DATA PLOTTED AGAINST VCO_2 DATA FOR ONE SCI SUBJECT DURING AN IET. BLACK LINES INDICATE THE CHANGE IN SLOPE AND DOTTED LINES INDICATE THE VO_2 AND VCO_2 POINTS WHERE CO_2 PRODUCTION EXCEEDED VO_2.

Subject	PIT	3 months	6 months	9 months	12 months
			Anaerobic Threshold ($L.min^{-1}$)		
1	0.18	0.29	0.31	0.30	0.29
2	0.33	0.17	0.38	0.34	0.4
3	0.13	0.21	0.19	0.18	0.2
4	0.21	0.24	0.36	0.33	0.36
5	0.36	0.34	0.34	0.43	0.39
Average	**0.24**	**0.25**	**0.32**	**0.32**	**0.33**
SEM	**0.10**	**0.07**	**0.08**	**0.09**	**0.08**
			Peak Respiratory Exchange Ratio		
1	0.92	1.13	1.20	0.99	1.13
2	0.84	0.91	0.92	1.00	0.97
3	1.02	1.19	1.07	1.14	1.28
4	0.81	1.13	1.05	1.05	1.13
5	1.26	1.09	1.10	1.08	1.14
Average	**0.97**	**1.09**	**1.07**	**1.05**	**1.13**
SEM	**0.08**	**0.05**	**0.05**	**0.06**	**0.05**
			Peak Blood Lactate Concentration (mmol)		
1	5.8	5.1	5.8	3.9	7.0
2	2.8	--	8.7	14.5	5.87
3	3.3	5.7	4.8	4.5	4.27
4	4.1	4.4	6.1	5.5	5.28
5	--	5.7	6.4	6.3	9.55
Average	**4.0**	**5.2**	**6.4**	**7.0**	**6.4**
SEM	**1.3**	**0.6**	**1.4**	**4.3**	**0.9**

TABLE 4.6: $\dot{V}O_2$ ($L.MIN^{-1}$) WHEN ANAEROBIC THRESHOLD WAS REACHED, PEAK RER AND BLOOD LACTATE CONCENTRATION (MMOL) FOR THE SUBJECTS OVER THE YEAR OF TRAINING.

Subject	PIT	3 months	6 months	9 months	12 months
Peak Rating of Perceived Exertion					
1	12.0	12.0	10.0	7.0	12.0
2	9.0	9.0	11.0	11.0	9.0
3	9.0	13.0	12.0	12.0	12.0
4	9.0	8.0	9.0	8.0	9.0
5	15.0	9.0	13.0	7.0	13.0
Average	**10.8**	**10.2**	**11.0**	**9.0**	**11.0**
SEM	**1.2**	**1.0**	**0.7**	**1.0**	**0.8**
Peak Rating of Perceived Breathlessness					
1	3.0	3.0	3.0	2.0	3.0
2	1.0	2.0	2.0	3.0	1.0
3	1.0	3.0	3.0	3.0	2.0
4	0.5	0.0	0.5	0.5	2.0
5	4.0	1.0	4.0	0.5	3.0
Average	**1.9**	**1.8**	**2.5**	**1.8**	**2.2**
SEM	**0.7**	**0.6**	**0.6**	**0.6**	**0.4**

TABLE 4.7: PEAK RATING OF PERCEIVED EXERTION AND PEAK RATING OF PERCEIVED BREATHLESSNESS FOR THE SUBJECTS OVER THE YEAR OF TRAINING.

4.4.2 CONSTANT LOAD EXERCISE TEST (CET)

Resting $\dot{V}O_2$ and V did not change significantly as a result of the training ($P > 0.05$).

As can be seen in Fig. 4.9, steady state $\dot{V}O_2$ had reduced significantly at 6 ($P < 0.01$), 9 and 12 ($P < 0.05$) months compared with post initial training and at 6 ($P < 0.01$) and 9 ($P < 0.05$) months compared with 3 month. Additionally, V reduced significantly at 6 and 9 months compared with post initial training and at 12 months compared with 3 months ($P < 0.05$).

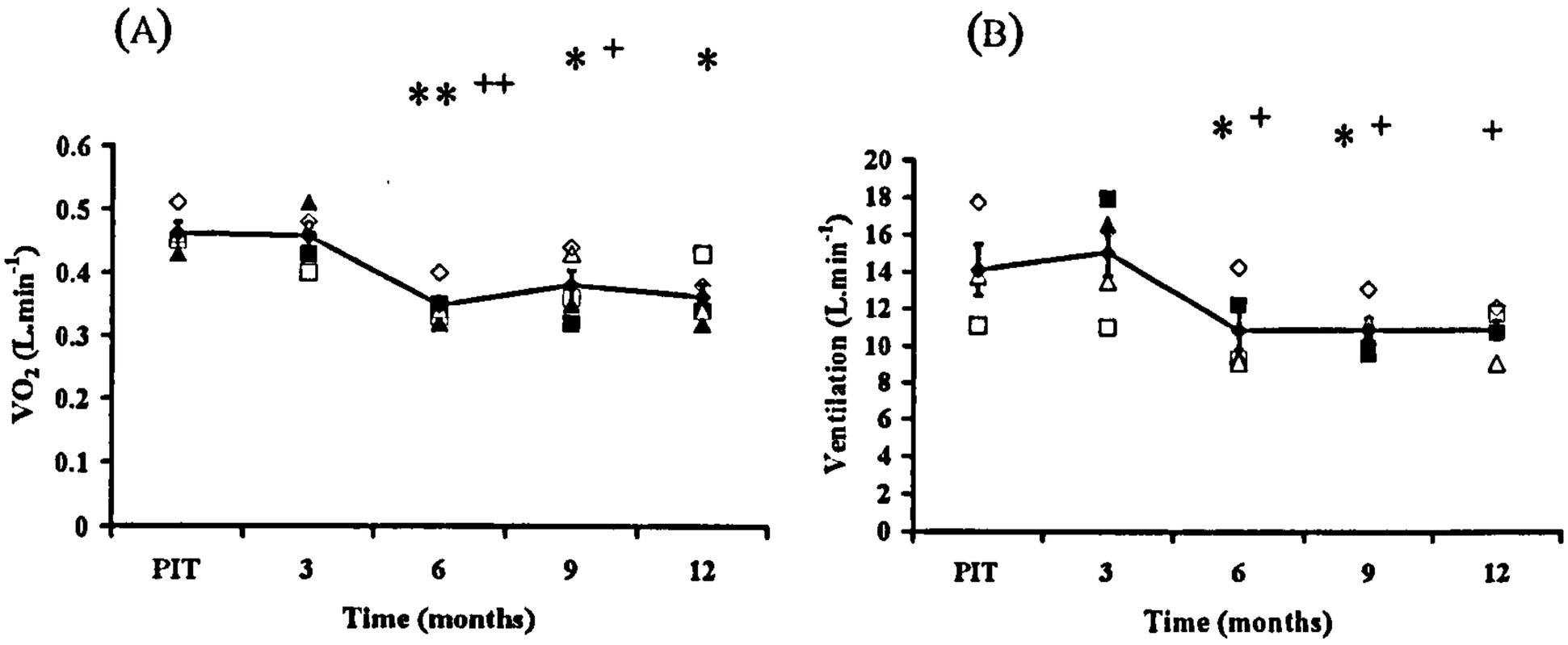

FIG. 4.9: AVERAGE STEADY STATE (A) OXYGEN UPTAKE ($\dot{V}O_2$) AND (B) VENTILATION (CLOSED DIAMOND AND LINE) AND INDIVIDUAL DATA (SUBJECT 1 CLOSED SQUARE, 2 OPEN SQUARE, 3 CLOSED TRIANGLE, 4 OPEN TRIANGLE AND 5 OPEN DIAMOND) OVER THE YEAR OF TRAINING. (SIGNIFICANTLY DIFFERENT TO POST INITIAL TRAINING *($P<0.05$) **($P<0.01$), SIGNIFICANTLY DIFFERENT TO 3 MONTHS $^{+}$($P<0.05$), $^{++}$($P<0.01$)).

Fig. 4.10 shows minute-by-minute relative $\dot{V}O_2$ values through out the entire CET at all time points. It can be seen that steady state $\dot{V}O_2$ decreased substantially at 6, 9 and 12 months compared with 0 and 3 months, but with no corresponding change in time to attain steady state and recovery time.

Work efficiency tended to improve throughout training (Fig. 4.11 a), although this did not attain statistical significance ($P=0.096$). Stimulation intensity showed a substantial decline through out the training programme (Fig 4.11 b), with 9 and 12 month data significantly reduced compared with post initial training ($P<0.05$). Additionally, stimulation intensity at 6, 9 ($P<0.01$) and 12 ($P<0.05$) months was significantly reduced compared with that at three months.

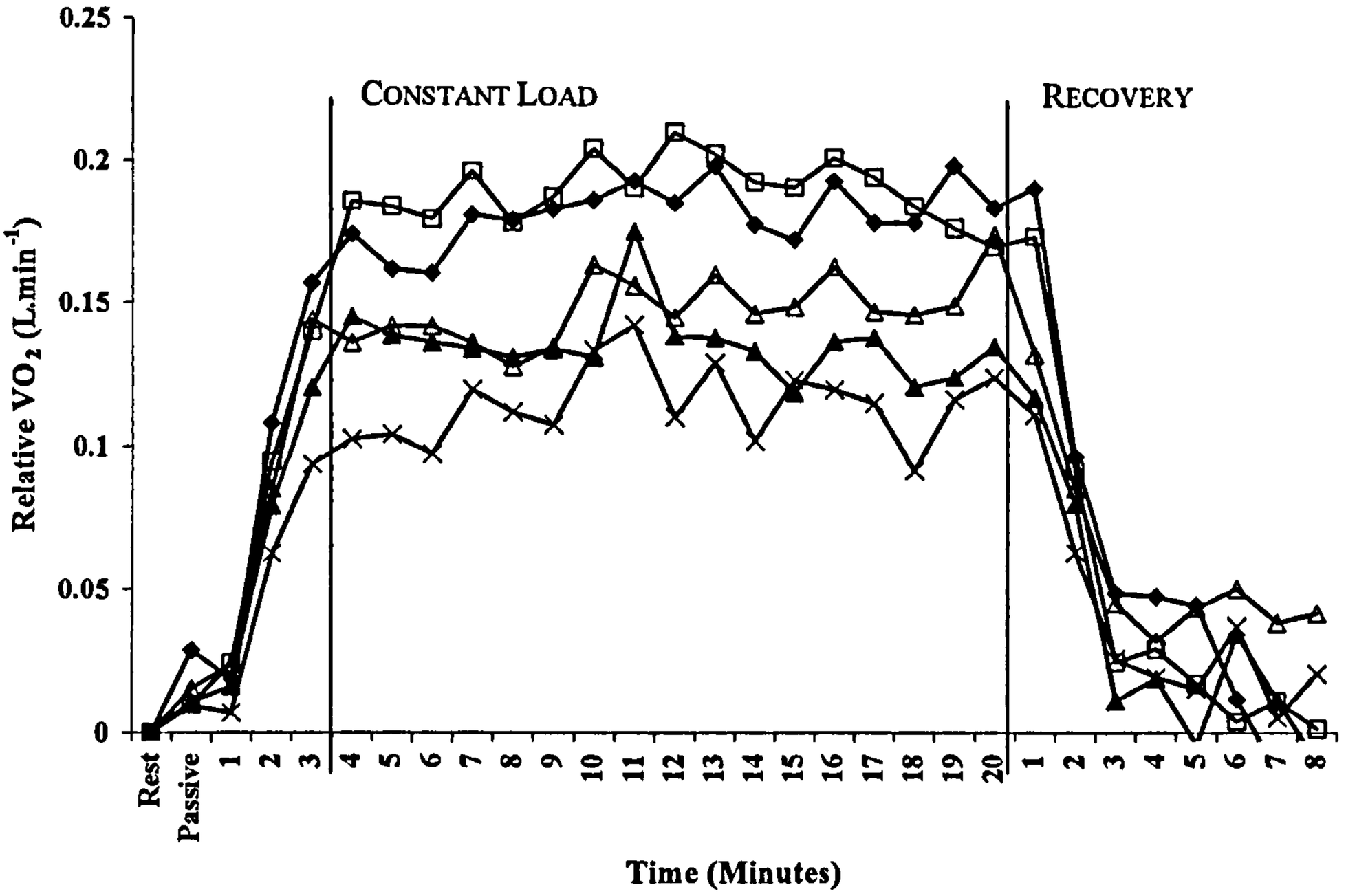

FIG. 4.10: AVERAGE OXYGEN UPTAKE ($\dot{V}O_2$) FOR 5 SUBJECTS DURING REST, PASSIVE, CONSTANT LOAD AND RECOVERY (LOWEST STIMULATABLE WORK RATE) PHASES OF A CONSTANT LOAD EXERCISE TEST AT POST INITIAL TRAINING (CLOSED DIAMONDS), 3 (OPEN SQUARES), 6 (CLOSED TRIANGLES), 9 (OPEN TRIANGLES) AND 12 (CROSS) MONTHS.

(A)

(B)

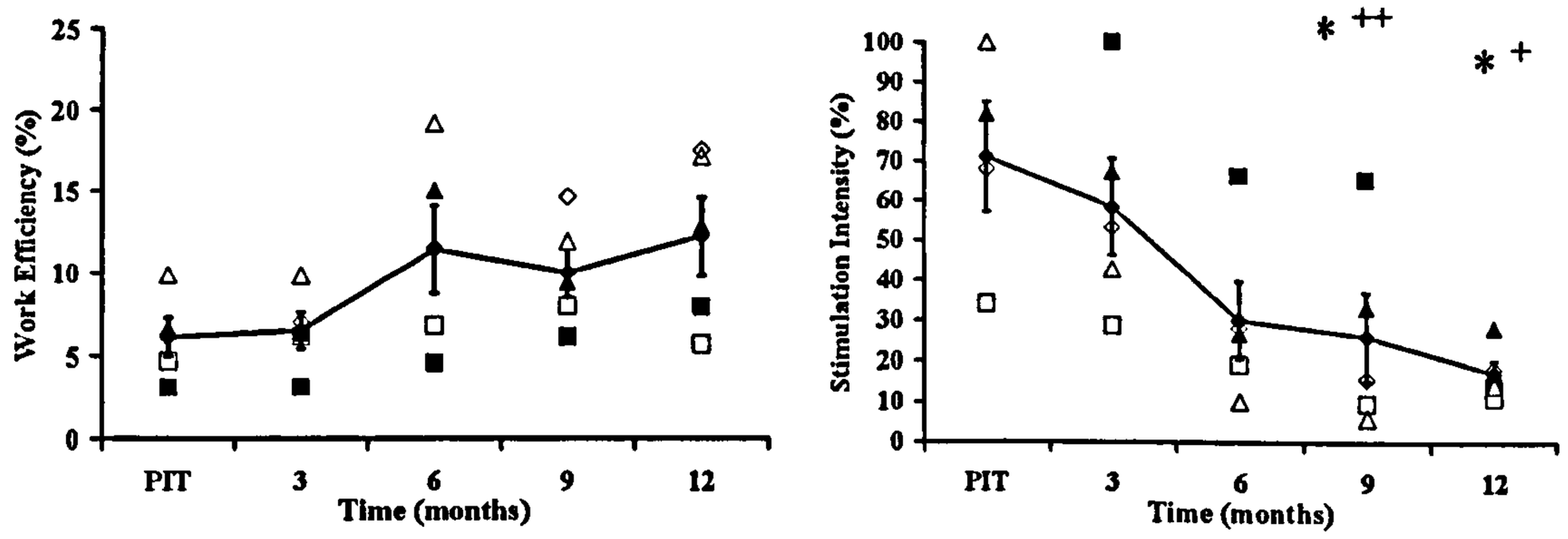

FIG. 4.11: AVERAGE STEADY STATE (A) WORK EFFICIENCY AND (B) STIMULATION INTESITY (CLOSED DIAMOND) AND INDIVIDUAL DATA (SUBJECT 1 CLOSED SQUARE, 2 OPEN SQUARE, 3 CLOSED TRIANGLE, 4 OPEN TRIANGLE AND 5 OPEN DIAMOND) OVER THE YEAR OF TRAINING (SIGNIFICANTLY DIFFERENT TO POST INITIAL TRAINING *($P < 0.05$), SIGNIFICANTLY DIFFERENT TO 3 MONTHS $^{+}(P < 0.05)$, $^{++}(P < 0.01)$.

There was no significant change in resting HR (P >0.05, Table 4.8), steady state HR, RER or peak lactate (P >0.05, Table 4.9). Rates of perceived exertion tended to decrease with time (Table 4.10) but this was not significant (P >0.05) and there was no concurrent change in rates of perceived breathlessness.

Subject	Heart Rate (bpm)				
	PIT	3 months	6 months	9 months	12 months
1	--	110.0	81.0	94.0	69.0
2	64.0	62.0	71.0	71.0	67.0
3	79.0	82.0	71.0	70.0	72.0
4	82.0	81.0	74.0	70.0	73.0
5	84.0	96.0	88.0	76.0	96.0
Average	**77.3**	**86.2**	**77.0**	**76.2**	**75.5**
SD	**9.1**	**18.0**	**7.4**	**10.3**	**11.9**

TABLE 4.8: STEADY STATE HEART RATE (BEATS PER MINUTE) AVERAGED OVER THE FINAL 10 MINUTES OF THE TEST FOR THE YEAR OF TRAINING.

Subject	PIT	3 months	6 months	9 months	12 months
Respiratory Exchange Ratio					
1	--	1.19	1.09	0.86	0.88
2	0.82	0.88	0.88	0.86	0.83
3	1.06	1.04	0.93	0.99	1.07
4	0.92	0.98	0.86	0.86	0.91
5	1.09	1.04	0.98	0.89	0.94
Average	**0.97**	**1.03**	**0.95**	**0.89**	**0.93**
SEM	**0.13**	**0.11**	**0.09**	**0.06**	**0.09**
Peak Blood Lactate Concentration (mmol)					
1	--	10.70	5.87	4.42	4.17
2	4.80	2.50	5.96	11.90	7.82
3	8.20	7.91	2.99	4.19	5.30
4	5.20	4.15	2.49	4.43	3.96
5	--	3.98	3.55	2.7	2.58
Average	**6.07**	**5.85**	**4.17**	**5.53**	**4.77**
SEM	**1.86**	**3.37**	**1.64**	**5.29**	**1.96**

TABLE 4.9: STEADY STATE RESPIRATORY EXCHANGE RATIO (RER) AND PEAK BLOOD LACTATE CONCENTRATION (MMOL) AVERAGED OVER THE FINAL 10 MINUTES OF THE TEST FOR THE YEAR OF TRAINING.

Subject	PIT	3 months	6 months	9 months	12 months
		Rates of Perceived Exertion			
1	--	12.0	10.7	7.0	7.7
2	7.0	7.0	7.0	7.0	7.0
3	11.0	12.0	9.0	10.3	11.0
4	9.3	7.3	7.0	6.7	7.0
5	10.3	6.0	6.0	6.0	6.0
Average	**9.4**	**8.9**	**7.9**	**7.4**	**7.7**
SEM	**1.7**	**2.9**	**1.9**	**1.7**	**1.9**
		Rates of Perceived Breathlessness			
1	--	3.5	2.7	1.2	1.2
2	0.5	0.5	0.7	0.0	0.7
3	1.0	2.7	1.0	1.3	2.0
4	0.5	0.2	0.3	0.0	0.0
5	0.8	0.0	0.0	0.0	0.0
Average	**0.7**	**1.4**	**1.0**	**0.5**	**0.8**
SEM	**0.2**	**1.6**	**1.1**	**0.7**	**0.8**

TABLE 4.10: STEADY STATE RATING OF PERCEIVED EXERTION AND RATING OF PERCEIVED BREATHLESSNESS OVER THE YEAR OF TRAINING.

4.5 DISCUSSION

4.5.1 INCREMENTAL EXERCISE TEST

Peak power output, $\dot{V}O_2$ and V increased significantly in response to the training programme. At 3, 6, 9 and 12 months of training peak power had increased by 27, 142, 192 and 234 % of post initial training respectively. Relative changes in peak $\dot{V}O_2$ were –0.9, 42, 47 and 66 % at 3, 6, 9 and 12 months, which were similar to the respective changes in V (12, 45, 51 and 69 %). There were no significant changes in peak heart rate, RER, lactate, RPE and RPB or anaerobic threshold.

In agreement with previous studies (Pollack et al., 1986; Pollack et al., 1989; Arnold et al., 1992; Goss et al., 1992; Hooker et al., 1992; Mohr et al., 1997; Mutton et al., 1997) peak power output increased significantly during the training. These changes were probably as a result of muscle hypertrophy since they significantly correlated with changes in thigh muscle depth (see Chapter 3) ($P < 0.05$, $R^2 = 63.2$ %, Fig. 4.6). Baseline data was taken at different time points for muscle depth (pre initial training) and power output (post initial training). Therefore the changes reported in muscle size after 3 months training probably occurred during the initial training phase. Both peak power and $\dot{V}O_2$ did not change significantly until post 6 months post training. In contrast, a number of other studies have noted significant improvements in peak power and $\dot{V}O_2$ after just 3 months training (Pollack et al., 1989; Arnold et al., 1992; Hooker et al., 1992). Considerable inter-individual variation in FES cycling ability and difficulty with training at the start of the cycling programme was found here and has been reported by others (Ragnarrson et al., 1988; Arnold et al., 1992; Mohr et al., 1997; Gerrits et al., 2000). Low subject numbers in this study might have resulted in a greater variability in early responses to training. Indeed, changes in power output following 3 months training were between -6 and 150 %, and 4 out of 5 subjects experienced only small improvements in power output during this period. Therefore the small improvement in $\dot{V}O_2$ may have been due to the small and variable changes in power output at the start of training.

The 42 % improvement in peak $\dot{V}O_2$ after 6 months training is greater than the 10-28 % improvement reported in other studies (Goss et al., 1992; Mutton et al., 1997). Mohr et al. (1997) reported a 19 % increase following a one year FES cycle training programme, which is lower than the 66 % improvement found after a year's training in this study. Cardiopulmonary adaptations to an FES cycling training programme involving high intensity training such as carried out here (one hour, 5 times per week) has not previously been reported. Training was carried out at a substantially lower frequency and duration (30 minutes, 3 times per week) in other studies (Goss et al., 1992; Mutton et al., 1997; Mohr et al., 1997), thus it is likely that greater adaptations took place in response to the greater training demand in this study.

Absolute $\dot{V}O_2$ values at peak power were 0.46 ± 0.06, 0.45 ± 0.06, 0.65 ± 0.06, 0.67 ± 0.08 and 0.76 ± 0.07 $L.min^{-1}$ at post initial training, 3, 6, 9 and 12 months respectively. These values are substantially lower than previously reported values of 0.78-1.30 and 0.95-1.43 $L.min^{-1}$ at pre and post training respectively (Goss et al., 1992; Hooker et al., 1992; Mohr et al., 1997; Mutton et al., 1997). The reason for this discrepancy is unclear. The main differences in previous studies compared with this one are that upright as opposed to recumbent cycling was used, different muscle groups were used, higher power outputs were attained and both tetraplegics and paraplegics were tested. The fact that different muscle groups were used is also unlikely to create a higher $\dot{V}O_2$ because fewer muscle groups were used in previous studies, which would theoretically result in smaller $\dot{V}O_2$ values. Since $\dot{V}O_2$ responses to FES exercise have been shown to be similar between paraplegics and tetraplegics

(Hooker et al., 1990; Faghri et al., 1992), it is unlikely that this variable caused the observed differences. It is possible that upright cycling is less efficient than recumbent cycling creating the higher $\dot{V}O_2$ values. Alternatively, the higher power outputs during upright cycling (10.5-55.0 W compared with 3.9-36.5 W) resulted in the greater demand for O_2 by the working muscles. Another contributing factor might be stimulation intensity, which is rarely reported.

The observed improvements in peak V and $\dot{V}O_2$ are due to either central or peripheral adaptations. The peak $\dot{V}O_2$ values attained were substantially lower than achieved by AB people exercising voluntarily at maximum intensity (3.0-5.0 $L.min^{-1}$). Additionally, higher peak $\dot{V}O_2$ values are attained when SCI people carry out voluntary arm exercise (1.2-1.4 $L.min^{-1}$) (Hooker et al., 1992; Mutton et al., 1997) or hybrid exercise (1.7-1.9 $L.min^{-1}$) (Mutton et al., 1997). Therefore it is unlikely that any central adaptations have taken place. In support of this, both peak ratings of perceived exertion and perceived breathlessness were substantially lower than would be expected for people working at maximum capacity (11 – Fairly light and 2 – Slight for RPE and RPB, respectively). Therefore it is likely that peripheral adaptations occurred. In support of this, substantial improvements in fatigue resistance during repetitive contractions of the quadriceps muscle occurred in the present study to levels comparable with AB people (Chapter 3). This indicates that peripheral adaptations such as improved capillarisation and increased mitochondrial density and aerobic enzymes of the citric acid cycle and electron transport chain occurred. Increased activity of glycolytic and mitochondrial oxidative enzymes have

been reported previously in response to a less intense FES cycle training programme (30 minutes, 3 times per week, Kjær et al., 2001a), which would result in a greater ability to metabolise fat and carbohydrates for energy production. Additionally the significant improvements in muscle size and strength (see Chapter 3) probably allowed SCI people to work at the higher peak power outputs after training. This would increase the peak O_2 demand by the muscles and thus $\dot{V}O_2$.

There were no significant changes in HR as a result of FES cycle training in agreement with previous studies (Mutton et al., 1997). However, other studies have shown significant increases in peak HR following a 3 month FES training programme (Pollack et al., 1989; Hooker et al., 1992). The lower power outputs attained in this study resulted in only small increases in HR as a result of the exercise. It is therefore unlikely that the exercise induced any substantial strain on the heart. Peak HR did tend to increase with power output, but remained low (81 ± 3.2, 86 ± 4.0, 89 ± 1.9, 90 ± 3.0 and 89 ± 5.3 bpm at post initial training, 3, 6, 9 and 12 months).

Peak RER and [lactate] were also unchanged throughout the training programme. Pollack et al. (1989) similarly showed no change in RER as a result of FES cycle training. In this study, VO_2 peak was defined as the point at which power output failed to increase, which appeared to occur when oxygen demand exceeded supply (RER > 1.0). At post initial training fatigue occurred at an average $\dot{V}O_2$ and RER of 0.46 $L.min^{-1}$ and 0.97 respectively. After one year training, fatigue occurred at a

significantly higher $\dot{V}O_2$ but at a similar RER and lactate. This suggests that improvements in muscle strength and fatigue resistance (see Chapter 3) allowed muscles to work aerobically for longer, thus higher peak power and $\dot{V}O_2$ were achieved.

4.5.2 CONSTANT LOAD EXERCISE TEST

Steady state $\dot{V}O_2$ and V decreased significantly at 6, 9 and 12 months compared with post initial training. There was also a substantial reduction in stimulation intensity required to maintain a steady state from 77 ± 12 % at post initial training to 58 ± 12, 30 ± 21, 26 ± 24 and 17 ± 6 % at 3, 6, 9 and 12 months, respectively. Work efficiency tended to improve throughout the training (Fig. 4.11), but this did not attain statistical significance. There was no significant change in steady state HR, RER, lactate, RPE or RPB as a result of the training.

Net $\dot{V}O_2$ ($\dot{V}O_2$ steady state - $\dot{V}O_2$ rest) values were 0.19 ± 0.03, 0.19 ± 0.03, 0.14 ± 0.02, 0.14 ± 0.02 and 0.12 ± 0.02 $L.min^{-1}$ at post initial training, 3, 6, 9 and 12 months respectively, indicating that a smaller uptake of oxygen was required to attain a similar power output post-training. This was unexpected because $\dot{V}O_2$ at any given work rate does not alter in AB people as a result of a training programme. In AB people it is unlikely that endurance training induces any substantial fibre type transformation although existing slow fibres would improve their aerobic capacity. Chronic SCI is known to result in a slow-fast fibre type transformation. It is

therefore likely that SCI people worked anaerobically at very low exercise intensities, utilising CHO as the predominant fuel source, resulting in higher $\dot{V}O_2$. It is possible that the training induced, to some extent, a fast-slow fibre type transformation. This would allow a greater aerobic contribution to exercise (due to increased fatty acid metabolism), which would result in reductions in $\dot{V}O_2$ at a given work rate as identified in the present study. However an associated reduction in RER during steady state exercise would be expected in this case, which was not observed in the present study (Table 4.9).

It has been reported in SCI people that a type IIx-a fibre type conversion takes place after ES training (Greve et al., 1993; Andersen et al., 1996; Crameri et al., 2002) and mRNA for the MHC I isoform is upregulated after only 2 weeks of ES (Harridge et al., 2002). Furthermore, Kjær et al. (2001a) reported significant improvements in glycolytic and mitochondrial oxidative enzyme activities following 3 months of FES cycle training. Mitochondrial density (Eisenberg & Salmons, 1981) and capillary:fibre ratio (Brown et al., 1976; Cabric et al., 1987; Nash et al., 1996) might also increase resulting in an improved ability to transport, take up and utilise oxygen. This would also result in a lower production or greater removal rate of lactate allowing an individual to work at higher intensities before the onset of blood lactate accumulation (OBLA).

Stimulation intensity at a given power output reduced significantly after training indicating that fewer motor units were required to attain a given power output. This was probably due to the significant improvements in muscle strength of the exercising muscles (see Chapter 3). It is therefore likely that the observed reduction in V and $\dot{V}O_2$ occurred in part because fewer motor units were active during steady state exercise after training.

RER values were 0.97 ± 0.06, 1.03 ± 0.05, 0.95 ± 0.04, 0.89 ± 0.03 and 0.93 ± 0.04 at post initial training, 3, 6, 9 and 12 months respectively. These values are higher than would be expected for AB people working voluntarily at similar work rates (Kjær et al., 1994) but are in agreement with previously reported values for this type of exercise in SCI people (0.99-1.02) (Pollack et al., 1989, Mohr et al., 1997). Similarly, lactate concentrations (2.6-11.9 mmol) were substantially higher than would be expected during voluntary exercise in AB people working at similar absolute levels (Kjær et al., 1994), indicating a relatively large anaerobic contribution. The greater proportion of fast twitch fibres in the paralysed muscles of SCI people would contribute to the high anaerobic contribution to exercise before training (Gerrits et al., 1999).

The training programme induced no significant changes in either RER or blood lactate concentrations, however RER tended to reduce (0.97 ± 0.13 and 0.93 ± 0.09 at post initial training and 12 months respectively). SCI people appeared to work anaerobically for long periods during their training (see Chapter 7) probably because

of the high stimulation intensity and frequency (50 Hz) used. In AB people there is a reduction in firing frequency during exercise in response to the contractile slowing and also additional motor units are recruited (De Luca et al., 1996; Adam et al., 2005). Therefore RER and [lactate] probably remained high even after training due to the stimulation parameters used.

There was however a tendency for both RER and [lactate] to decrease throughout the programme (Table 4.9). As mentioned previously, FES cycling is known to result in a type IIx-a conversion (Greve et al., 1993; Andersen et al., 1996; Crameri et al., 2002) and mRNA for the MHC I isoform is upregulated after ES training (Harridge et al., 2002). Thus a fibre type conversion might have taken place as well as increases in mitochondrial density and oxidative enzymes, increasing the capacity for fat metabolism and therefore reducing RER and [lactate] at a given work rate. Alternatively, the anaerobic-type training might have improved the body's capacity for acid-base regulation (chemical buffering mechanisms and respiratory elimination of CO_2 to control pH), which would reduce the detrimental effects of anaerobic exercise. However little evidence exists to suggest the occurrence of such adaptations.

Work efficiency is a measure of the percentage of the total energy expended that contributes to work. In AB people, work efficiency during stationary cycling is approximately 20–39 % with the remaining energy lost as heat. As shown in Fig. 4.11 (a), work efficiency tended to improve with training but this did not attain

statistical significance (P = 0.096). Work efficiency was very low at the start (6.1 ± 1.1 %) and improved by 100 % after 12 months training (12.2 ± 2.4 %) but remained low compared with AB people. It is not expected that work efficiency would change substantially in AB people as a result of a training programme. It is likely that efficiency tended to improve here due to the reductions in $\dot{V}O_2$ at a given work rate. It is also likely that other variables contributed to the reduced efficiency during this type of exercise, causing the data to be more variable. Such factors include higher than normal proportions of fast twitch fibres even after training, high frequency stimulation and synchronous recruitment of motor units, which would all result in a greater anaerobic contribution to exercise. Furthermore, diet was not controlled over a one year period and any changes might have influenced efficiency by affecting metabolism during exercise. Variations in resting $\dot{V}O_2$ might also have affected work efficiency values since the use of a mask for measuring $\dot{V}O_2$ might have induced over-breathing during rest and/or exercise.

There were no effects of training on HR (Table 4.5). Therefore there was no stimulus for cardiac hypertrophy and consequently submaximal HR at a given power output did not change substantially. In contrast, improvements in HR, stroke volume and cardiac output have been reported following a short term FES training programme (Faghri et al., 1992), which suggests improvements in central cardiovascular fitness. It is possible that the higher power outputs achieved in that study placed a relatively greater demand on the central circulatory system resulting in central cardiovascular adaptations.

Both RPE and RPB were low throughout the training period (Table 4.10) and did not change substantially as a result of the training. Since the CET induced little stress on the cardiopulmonary system, with small effects on HR (Table 4.8) and breathing frequency (data not presented), post initial training RPE and RPB were low, leaving little potential for change.

4.6 Methodological Limitations

There are a number of limitations that should be considered when using standard exercise tests to assess cardiopulmonary fitness in SCI people using FES cycling. AB people carrying out $\dot{V}O_2$ max testing are required to attain volitional exhaustion and a plateau of HR. These criteria were not attained during IET testing in SCI people and HR, RPE and RPB remained very low. This indicates that exclusively peripheral factors induced fatigue. It is therefore possible that the improved power output after training (due to muscular adaptations) resulted in the increased oxygen demand and therefore $\dot{V}O_2$ observed after training. Therefore it is unlikely that improvements in cardiovascular fitness that would result in reduced risk of disease occurred. Additionally, controls were not used in this study and therefore it was not possible to identify changes that occur over a one-year period when no intervention is employed.

It should finally be noted that very small subject numbers were used in this study and data to assess the intra-subject variability in these tests was not collected.

Therefore it cannot be discounted that the changes observed here were less than the co-efficient of variation of the technique itself.

4.7 CONCLUSIONS

Peak power output, $\dot{V}O_2$ and V improved significantly after 6 months training. These changes were probably due to peripheral adaptations including muscle hypertrophy, improved capillarisation and increased mitochondrial density and aerobic enzymes of the citric acid cycle and electron transport chain resulting in a greater ability to metabolise fat and carbohydrates for energy production. However, peak power output $\dot{V}O_2$ and V remained substantially lower than would be expected from AB people exercising voluntarily. A long-term intensive training programme did result in greater relative changes in peak oxygen uptake compared with previous training studies. Absolute values were however lower than previously reported due possibly to differences in the cycle positioning in this study.

Submaximal $\dot{V}O_2$ and V reduced significantly as a result of the training, which might be a result of a fast-slow fibre type transformation. The reduction in stimulation intensity for a given power output indicates that fewer motor units were active to attain the steady state work rate, which might also have contributed to the observed reductions in $\dot{V}O_2$ and V. Work efficiency tended to improve with training but this did not attain statistical significance probably because higher proportions of fast twitch fibres, high frequency stimulation and synchronous recruitment of motor units limited efficiency. Peak and steady state RER and [lactate] were higher than

expected and did not change as a result of the training suggesting a high anaerobic contribution to exercise even after a one year training programme.

Overall, adaptations as a result of the training programme appear to have improved aerobic capacity in SCI people in that their capacity for aerobic ATP regeneration improved as indicated by improvements in peak power output and $\dot{V}O_2$ and reduced $\dot{V}O_2$ at a given work rate. It should however be noted that $\dot{V}O_2$, V, RPE and RPB were low compared with AB people and thus were unlikely to be limiting factors in ATP generation. Thus it is unlikely that central cardiopulmonary adaptations occurred, which is further supported by the fact that resting and submaximal HR did not change due to the training. Presumably peripheral adaptations that resulted in improved muscle strength and fatigue resistance allowed a higher power output and therefore $\dot{V}O_2$ to be attained during the IET.

Although cardiovascular changes were small it is possible that the training programme did result in improvements in health since it has been reported that an increase in weekly energy expenditure of 4200 kilojoules reduces mortality risk by ~20 % (Warburton et al., 2006). Based on efficiency data during FES cycling (CET at 70 % post initial training peak power) SCI people expended on average 20.8 kilocalories per minute. Based on the assumption that all training was carried out at this intensity, the average increase in weekly energy expenditure would be 6240 kilojoules indicating a substantial reduction in mortality risk due to training.

The following chapter considers the effects of the year long FES training programme on pressure sore susceptibility (gluteal muscle bulk, peak seating pressures and tissue oxygenation) for SCI people.

Chapter 5 Tissue Oxygenation and Seating Pressures.

The work presented in this chapter aimed to assess the effects of a one year FES cycling training programme on measures of pressure sore susceptibility in SCI people. This was assessed in three ways i) transcutaneous oxygen pressure at the sacrum and the response to mechanical loading ii) peak seating pressures in a standard NHS wheelchair and each subjects' own wheelchair and iii) gluteal muscle size and muscle and subcutaneous tissue thickness under the ischea.

5.1 Literature Review

Pressure sores are a common complication for people with spinal cord injury (SCI). Reduced mobility combined with muscle wastage around bony prominences (Taylor et al., 1993; Round et al., 1993) mean that SCI people are exposed to long periods of potentially pathologically high pressures around their ischial tuberosities and sacrum (Kabagambe et al., 1994; Aissaoui et al., 2001). Reduced blood flow below the lesion level (Taylor et al., 1993) and reduced integrity of the microcirculation to remove metabolic waste products (Krouskop et al., 1978; Krouskop, 1983) are often important factors that contribute to the possible development of pressure sores.

There are two main ways in which increased exercise of paralysed muscles might reduce the susceptibility of pressure sores: 1) improved microcirculation enhancing tissue oxygenation and improving recovery from imposed loads including the removal of metabolic waste products, and 2) improved muscle bulk in the gluteal region reducing peak seating pressures around the ischial tuberosities.

5.1.1 MICROCIRCULATION

Capillary growth occurs in response to physical conditioning in AB people (Andersen & Henriksson, 1977) and in response to prolonged low frequency ES in animal models (Brown et al., 1976; Hudlicka et al., 1977) and AB humans (Salmons & Henriksson, 1981; Pérez et al., 2002). Twenty eight days of CLFS increased resting and exercising blood flow in animal muscle (Hudlicka et al., 1977) and resistive vibration exercise attenuated the reduction in the diameter of the common femoral artery during bed rest in AB people (Bleeker et al., 2005). These adaptations are possibly induced by increased blood flow and arterial shear stress in response to the increased demand for oxygen by the active muscles (for review see Salmons & Henriksson, 1981; Hudlicka et al., 1992).

An increase in muscle blood flow occurs in SCI people during ES above the motor threshold (Levine et al., 1990b; Kim et al., 1995; Scremin et al., 1998; Olive et al., 2002) and has been reported to be similar in magnitude to that of AB people exercising with ES at similar workloads (Olive et al., 2002). Thus it is possible that angiogenesis would occur in SCI people in response to ES training. A short-term programme of FES cycling has been reported to induce increases in the cross sectional area of the femoral artery and improve blood flow in this population (Gerrits et al., 2001a; Thijessen et al., 2005). This effect was localised to the stimulated musculature (Thijessen et al., 2005). The number of capillaries surrounding each fibre increased significantly in SCI people following a 10-week FES cycling training programme consisting of three, 30 minute sessions per week (Crameri et al., 2002). Kano et al. (1997) noted that capillary:fibre ratio

significantly increased in animal muscle following both a low and high intensity training programme, although they resulted in reductions and increases respectively in capillary lumen density.

It has been reported that training status has no effect on reactive hyperaemia in AB people (Boegli et al., 2003). In tetraplegics however, Nash et al. (1996) reported that exercise trained people had an improved hyperaemic response to occlusive challenge compared with untrained people (Nash et al., 1996). Thus the improved microcirculation resulting from ES induced exercise might induce enhanced or more rapid recovery from blood flow occlusion.

5.1.1.1 Tissue Oxygenation

The implications of the above for oxygenation of the skin and subcutaneous tissue are unclear (Levine et al., 1990b). An increase in cutaneous blood flow has been reported in SCI people during ES above the motor threshold. It has also been noted in AB people that a significant increase in skin O_2 levels occurs 30 minutes after ES when the stimulus creates a muscular contraction (Baker et al., 1983). ES below the motor threshold had no effect on skin oxygenation (Baker et al., 1983). Kett et al. (1988) reported that gluteal muscle ES increased blood flow with a trend towards increased skin blood flow, although statistical significance was not achieved, probably due to low subject numbers (Bogie et al., 2000).

It has been observed that ES of the gluteal muscles during sitting in SCI people results in local increases in T_CPO_2, which cease with the removal of ES (Bogie et al., 2000). High voltage (>100 volts) pulsed galvanic stimulation has also been

shown to increase T_CPO_2 at the sacrum from 49 to 66 mmHg (Mawson et al., 1993). Conversely, Gilcreast et al. (1998) found that T_CPO_2 significantly decreased in response to ES of the foot in a group of diabetic patients. The inconsistency in these studies might be due to variations within subject groups. Gilcreast et al. (1998) identified subgroups of responders and non-responders to ES, with the responders being people with a high risk for wound-healing problems. Mawson et al. (1993) similarly noted that the greatest increases in T_CPO_2 were observed in the subjects with T_CPO_2 values at or below the normal range (60-100 mmHg).

An 8 week programme of FES standing (ES of vastus lateralis, gluteus maximus, hamstrings and erectus spinae) has been reported to result in an insignificant increase in baseline tissue oxygenation values at the ischium in most, but not all, SCI people (Bogie & Triolo, 2003). No correlation between % increase in T_CPO_2 and exercise time could be established (Bogie & Triolo, 2003). There was a large range of T_CPO_2 values (44.5-98.6 and 43.7-88.5 at pre- and post-training respectively). This might be due to variability between and/or within subjects or might reflect poor repeatability in the technique used and may have disguised any changes that occurred as a result of the training. It is therefore important to determine inter- and intra-subject variability of this methodology in AB people in order to determine its sensitivity.

To my knowledge, there has been no investigation to identify the effect of a long-term ES programme on tissue oxygenation in SCI people. It is possible that this would result in improved microvasculature, which would provide an

important enhancement in the supply of O_2 and essential nutrients to cutaneous tissue in paralysed parts. This might enable SCI people to withstand the detrimental effects of seating pressures for longer periods, recover from the local tissue hypoxia more effectively and be less susceptible to pressure sores.

5.1.2 SEATING PRESSURE AND GLUTEAL SIZE

It is well known that a period of ES exercise results in improved muscle bulk in SCI people (Petrofsky, 1987; Pacy et al., 1988; Arnold et al., 1992; Hjeltnes et al., 1997; Mohr et al., 1997; Scremin et al., 1999; Crameri et al. 2002; Sköld et al., 2002). Thus ES of the gluteal muscles, resulting in improved muscle bulk around bony prominences might reduce the high peak pressures that have been reported around the ischeal tuberosities of SCI people (Thorfinn et al., 2002; Gutierrez et al., 2004) due to muscle wastage.

Bogie & Triolo (2003) reported that an 8 week programme of FES standing (ES of vastus lateralis, gluteus maximus, hamstrings and erectus spinae) resulted in no significant change in overall mean interface pressure measured in an individuals' own wheelchair. Mean ischeal region pressures however reduced significantly after exercise (Bogie & Triolo, 2003). It is therefore likely that hypertrophy of the gluteal musculature resulted in pressures being distributed over a larger surface area, thus reducing peak pressures around the bony prominences.

It has been shown that maximum seating pressures around the ischeal tuberosities are significantly reduced and redistributed to the surrounding areas

during ES in both AB and SCI people (Levine et al., 1989; Ferguson et al., 1992; Bogie et al., 2000). Levine et al. (1990a) noted that low level ES of the gluteal muscles during sitting results in changes in the shape of the buttocks to more closely resemble buttocks under no load. Therefore, ES has the potential to serve as an alternative to pressure lifts, which might reduce susceptibility to shoulder injury.

5.2 T_CPO_2 RELIABILITY

This reliability study aimed to identify inter and intra reliability of resting T_CPO_2 measurements and recovery time after a mechanical insult in AB people.

5.2.1 INTRODUCTION

Transcutaneous oxygen pressure (T_CPO_2) measurements are a non-invasive and indirect method of measuring arterial oxygen tension (Huch et al., 1972) and can be utilised to indicate the ability of the microcirculation to respond to metabolic demand from surrounding tissues (Newson et al., 1981; Newson & Rolfe, 1982). The methodology was initially developed as a non-invasive measure of arterial PO_2 in neonates (Huch et al., 1972) and is now used as a research tool to assess tissue oxygenation in people at risk of pressure sore development. There has however been little research investigating T_CPO_2 in AB people.

The technique measures surplus O_2 at an artificially elevated skin temp (≈43/44 °C). This creates pressure gradient, allowing O_2 to descend from the dermis through epidermis and, at the stratum corneum, continues to a layer of contact

gel and the face of the sensor. Cutaneous oxygen (O_2) values were indicative of tissue viability by reflecting the arterial blood flow changes in response to load (Swain & Grant 1989). The measurement is affected by local capilliarisation, blood flow and tissue oxygen consumption, causing day to day variations in T_cPO_2. Measurements are taken from small areas of the skin that are not necessarily representative of the surrounding area.

Previous work has shown intra-individual variations in T_CPO_2 of at least ± 7mmHg at given anatomical sites (Olerud et al., 1987; Daviet et al., 2004). Rooke & Osmundson (1989) reported at least a ± 23.5 % variation in T_CPO_2 at two adjacent sites in the foot. Coleman (1986) reported an average intra-individual variation in T_CPO_2 of approximately 10 % but pointed out that this was not significant when compared with T_CPO_2 levels that reflect skin ischemia (40 mmHg). T_CPO_2 measurements have been said to be unsuitable for the assessment of hemiplegics with microcirculatory disorders because daily variations in T_CPO_2 are greater than any shift caused by the disorder itself (Daviet et al., 2004).

Bader and Gant (1985) studied the effect of external loading on T_CPO_2 in healthy young and elderly subjects. They reported considerable variation in the level of tolerable pressure in both groups, but noted a characteristic pattern between T_CPO_2 and applied pressures. In response to provocation, such as local hyperthermia or reactive hyperaemia, relative changes in T_CPO_2 values appear to be more discriminating than absolute values (Diamantopoulos et al., 1995).

No significant differences in T_CPO_2 have been shown between SCI and able-bodied people (Patterson et al., 1993; Kabagambe et al., 1994; Peters, 2000). However, the rate of recovery after the removal of an external load may be an impaired in SCI people (Kabagambe et al., 1994).

This reliability study aimed to establish the intra-individual variation in T_CPO_2 under known loads and relative changes during recovery after the removal of load in AB people. It is important to understand these day to day variations in order to be able to establish a discriminative test that can identify impairments in the microcirculation of SCI people.

5.2.2 METHODOLOGY

Two experiments were carried out. The first investigated the reliability of T_CPO_2 values in response to increasing known mechanical loads until T_CPO_2 fell <20 mmHg and also the recovery time of T_CPO_2 following the removal of load. The second investigated the reliability of T_CPO_2 recovery time following the removal of a single known mechanical load. These two separate experiments were carried out in order to investigate whether recovery from a load that reduces T_CPO_2 to <20 mmHg was more or less reliable than recovery from a single known load. Five healthy subjects (4 female) carried out both experiments. Mean (± SEM) age and body mass index were 36 ± (5.2) years and 22.1 ± (1.6) respectively. Each subject was tested on 5 separate occasions for each experiment (10 tests per subject in total).

The experimental set-up consisted of a balanced beam with a moveable weight at one end. This counterbalanced a loading pan directly above a rigid indentor head, that incorporated the transcutaneous oxygen electrode (Bader & Gant, 1985; Bader & Gant, 1988; Bader, 1990). All measurements were carried out on the lower back at the sacrum. Subjects were positioned lying prone with a pillow underneath the lumbar to ensure that the indentor head could be placed on a flat area of skin in the sacral spine region (Fig. 5.1).

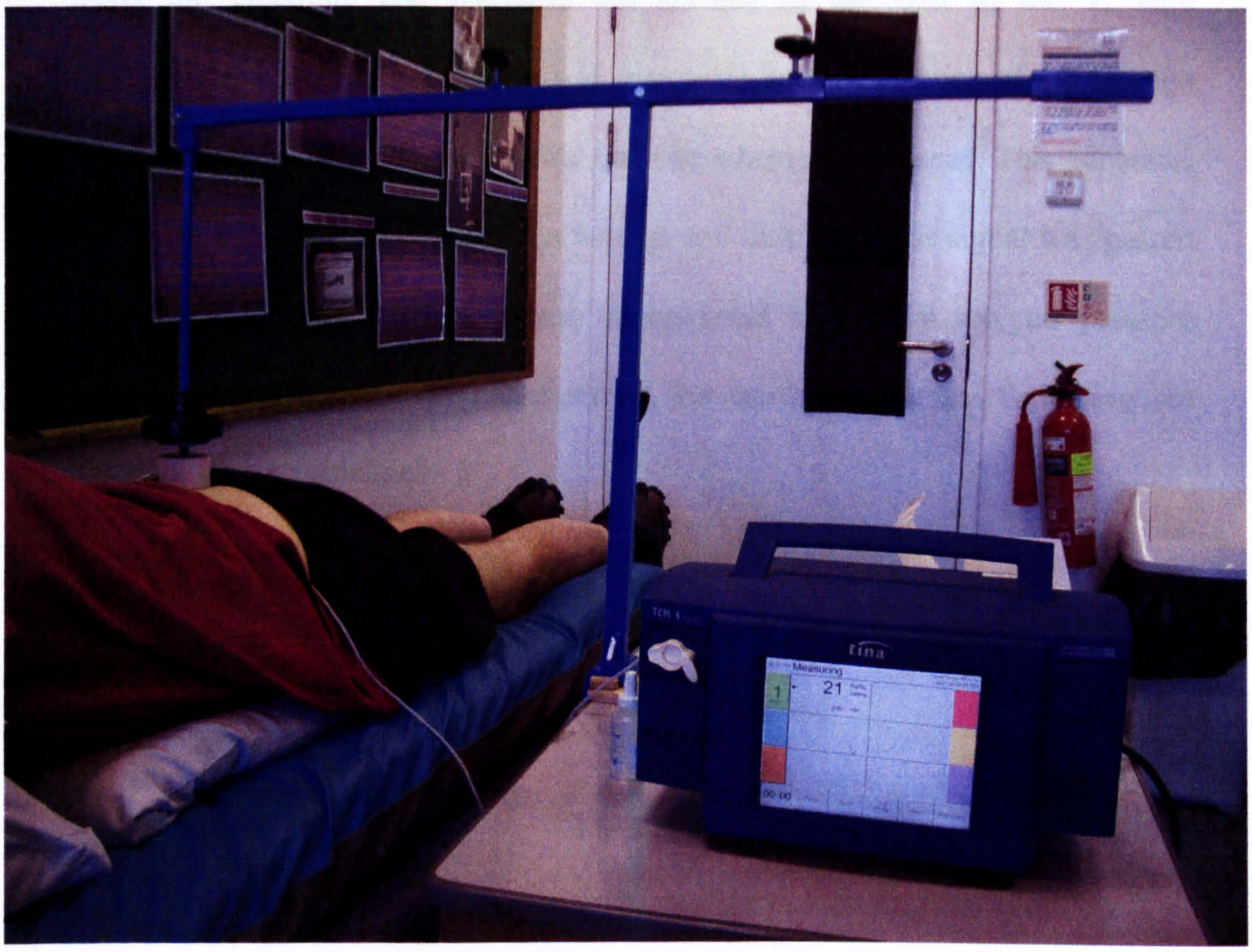

FIG. 5.1: BALANCED BEAM WITH MOVABLE WEIGHT AT ONE END. T_CPO_2 ELECTRODE IS ENCASSED WITHIN THE CYLINDER ATTACHED AT THE SACRUM. DIRECTLY ABOVE THIS IS THE LOADING PAN CONTAINING WEIGHTS.

The skin was cleaned with an alcohol swab and allowed to air-dry. An attachable encasing was then fixed to the skin and filled with electrolyte solution. The electrode was covered with electrode solution and a membrane ensuring there

were no air bubbles present. It was encased in a cylinder (6 mm diameter) and encased within the indentor head (45 mm diameter). The electrode was then attached to the encasing fixed to the skin and the moveable weight was adjusted so that the indentor head was only just touching the skin, balanced with minimal pressure on the skin.

The electrode combines a heating element, temperature sensors and a Clake-type oxygen electrode (TCM400, Radiometer, Copenhagen). The heating element heats the electrode and therefore the surface of the skin to 44 °C with thermostatic control. O_2 pressure was measured by the diffusion of O_2 through the membrane of the electrode to the cathode where it is reduced. This generates a current, which is converted into a voltage and digitised. This signal was passed to a microcomputer where it was reconverted to display oxygen pressure (mmHg). The system was calibrated at the start of each test following the manufacturer's guidelines.

At the start of each test a period of 20 minutes was given to allow equilibrium to be attained, and the baseline T_CPO_2 value was recorded.

5.2.2.1 Experiment 1: T_CPO_2 responses to mechanical loading and recovery.

After the baseline T_CPO_2 value had been attained a weight of 500 grams (g) was added to the loading pan and T_CPO_2 was recorded after a period of 5 minutes. 200 g weights were then progressively added to the loading pan at 5 minute intervals. T_CPO_2 was recorded at the end of each 5 minutes of load. When T_CPO_2 fell to <20 mmHg the load was removed. T_CPO_2 was recorded every 20 seconds

for 3 minutes during recovery and was expressed as a percentage of the baseline T_CPO_2.

5.2.2.2 Experiment 2: T_CPO_2 recovery following the removal of a known mechanical load.

After the baseline $TcPO_2$ value had been attained a load of 3000 g was applied to the loading pan for a period of 2 minutes. This load was chosen because the radius of contact of the indenter is 22.5 mm, giving a pressure of 42 mmHg per 1000 g of load. 3000 g would therefore give a pressure of 126 mmHg (~ systolic blood pressure) and thus should reduce T_CPO_2 to ~0 mmHg. After 2 minutes the load was removed and T_CPO_2 was recorded every 20 seconds for 3 minutes. T_CPO_2 during recovery was expressed as a percentage of the baseline T_CPO_2.

5.2.3 DATA ANALYSIS

Mean, standard error of measurement (SEM) and coefficient of variation (CV) of baseline T_CPO_2 and T_CPO_2 three minutes after the removal of load for the 5 trials were calculated for each subject. The same values were calculated for the 50 % recovery time during the 5 trials for each subject and across the 5 subjects for each trial to assess intra- and inter-subject variability respectively. Paired Student T-Tests were used to compare pre and post load T_CPO_2 values within each experiment and between experiments.

5.2.4 RESULTS

5.2.4.1 Experiment 1: T_CPO_2 responses to mechanical loading and recovery.

Average baseline T_CPO_2 was 68.3 ± 2.3 mmHg with a CV of 7.43 %. Three minutes after load had been removed, average T_CPO_2 was similar to baseline (68.4 ± 4.6 mmHg) with a CV of 14.23 % (Table 5.1).

Subject	Before load TcPO2 (mmHg)			After load TcPO2 (mmHg)		
	Mean	SEM	CV	Mean	SEM	CV
1	62.0	1.38	4.97	77.8	5.93	17.04
2	76.2	3.48	10.22	65.0	6.91	23.76
3	64.3	3.03	10.60	61.3	4.70	13.28
4	67.6	2.29	7.59	67.8	3.07	10.13
5	71.6	1.21	3.77	70.1	2.16	6.93
Mean	**68.28**	**2.28**	**7.43**	**68.38**	**4.56**	**14.23**

TABLE 5.1: TRANSCUTANEOUS OXYGEN PRESSURE (T_CPO_2) BEFORE AND AFTER LOAD FOR 5 PEOPLE ACROSS 5 TRIALS.

The load required to cause T_CPO_2 to fall to <20 mmHg varied from 700-1500 g across all trials. Ranges for the 5 individual subjects were 700-1500, 700-1300, 700-1500, 700-1300 and 700-1300 g across the 5 trials (Table 5.2).

		Subject				
		1	2	3	4	5
Load (g)	Trial 1	1100	700	900	1300	1100
	Trial 2	700	1300	1300	700	700
	Trial 3	1500	1300	1500	1300	700
	Trial 4	700	1300	700	1100	900
	Trial 5	700	900	1100	900	1300
	Mean	**1100**	**940**	**1100**	**1060**	**940**
	SEM	**283**	**358**	**316**	**241**	**261**
	CV	**25.7**	**38.1**	**28.7**	**24.6**	**27.7**

TABLE 5.2: LOAD REQUIRED TO REDUCE T_CPO_2 TO <20 MMHG FOR 5 PEOPLE ACROSS 5 TRIALS.

T_CPO_2 responses with loads up to 1500 g or causing T_CPO_2 to fall <20 mmHg if that occurred before 1500 g are shown in Fig. 5.2. T_CPO_2 ranged from 24-61, 5-59, 10-62, 1-54, 4-39 and 8-19 mmHg for individual subjects across 5 trials under loads of 500, 700, 900, 1100, 1300 and 1500 g, respectively. Intra subject CV ranged from 13.1-36.8, 44.0-69.6, 0.0-56.7, 22.6-133.3 and 54.4-73.3 % after 5 minutes under loads of 500, 700, 900, 1100 and 1300 g, respectively (Table 5.3). Time taken for recovery across 5 trials is shown in Fig. 5.3.

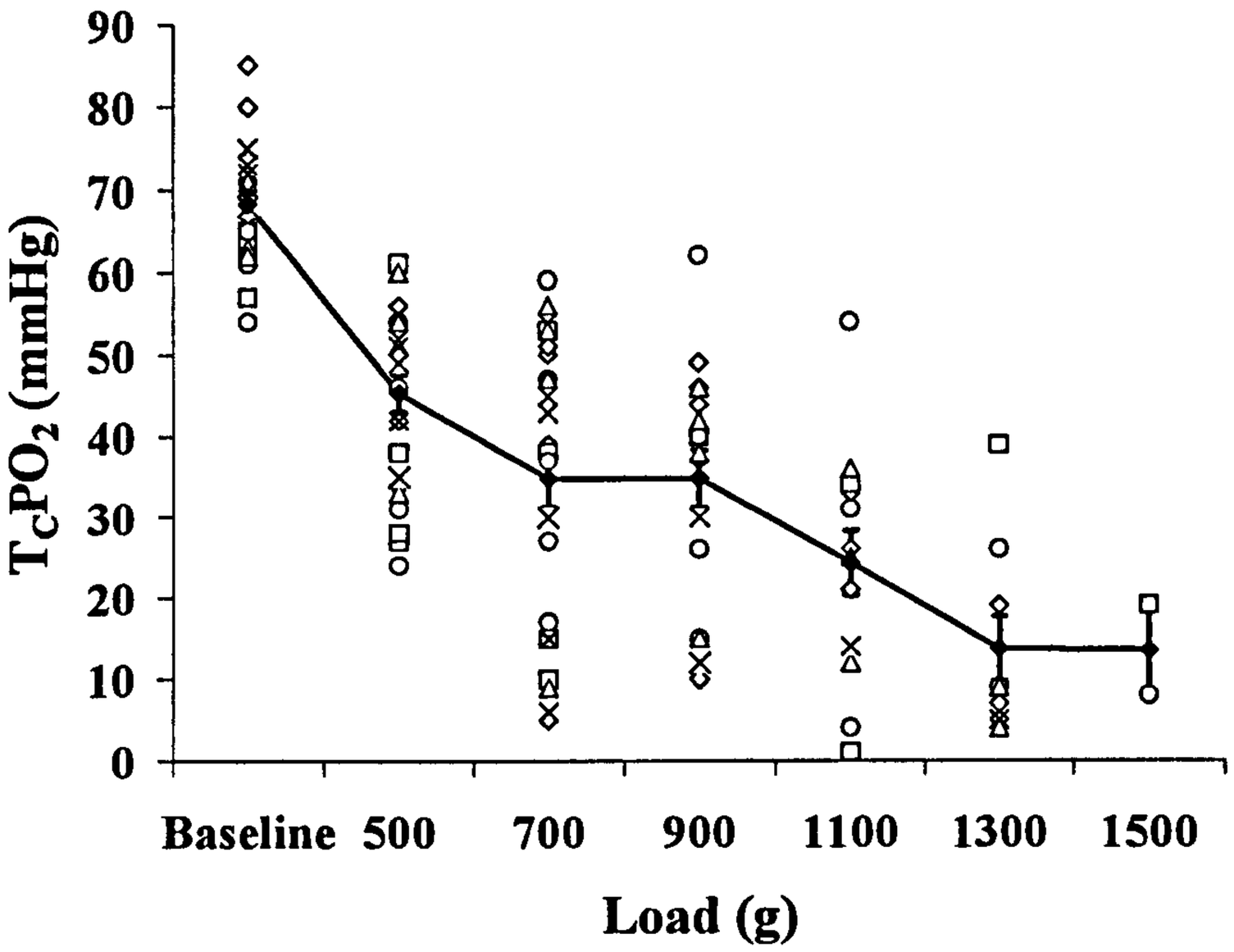

FIG. 5.2: TRANSCUTANEOUS OXYGEN PRESSURE (T_CPO_2) AT BASELINE AND IN RESPONSE TO 5 MINUTES UNDER DIFFERENT LOADS. AVERAGE DATA (MEAN ± SEM, CLOSED DIAMONDS AND SOLID LINE) AND INDIVIDUAL DATA (1 = OPEN DIAMONDS, 2 = OPEN SQUARES, 3 = OPEN CIRCLES, 4 = OPEN TRIANGLE AND 5 = CROSS).

		500 g	700 g	900 g	1100 g	1300 g	1500 g
Subject 1	T_cPO_2 (mmHg)	53.2	40.0	37.3	26.7	10.3	--
	SEM	3.1	9.14	9.1	3.5	4.4	--
	CV (%)	13.1	51.1	49.1	22.6	73.3	--
Subject 2	T_cPO_2 (mmHg)	37.6	26.4	40.0	17.5	39.0	19.0
	SEM	6.2	8.2	0.0	16.5	--	--
	CV (%)	36.8	69.6	0.0	133.3	--	--
Subject 3	T_cPO_2 (mmHg)	40.2	37.4	35.8	29.7	17.5	8.0
	SEM	5.5	7.4	10.1	14.5	8.5	--
	CV (%)	30.6	44.0	56.7	84.4	68.7	--
Subject 4	T_cPO_2 (mmHg)	49.4	42.4	35.3	24.3	6.5	--
	SEM	4.5	8.5	6.9	6.9	2.5	--
	CV (%)	20.4	45.0	39.4	49.4	54.4	--
Subject 5	T_cPO_2 (mmHg)	46.4	27.8	26.0	19.5	5.0	--
	SEM	3.5	7.7	7.2	5.5	--	--
	CV (%)	17.1	61.5	48.0	39.9	--	--

TABLE 5.3: T_cPO_2 UNDER EACH LOAD FOR 5 SUBJECTS ACROSS 5 TRIALS (-- NO VALUE DUE TO T_cPO_2 FALLING TO <20 MMHG).

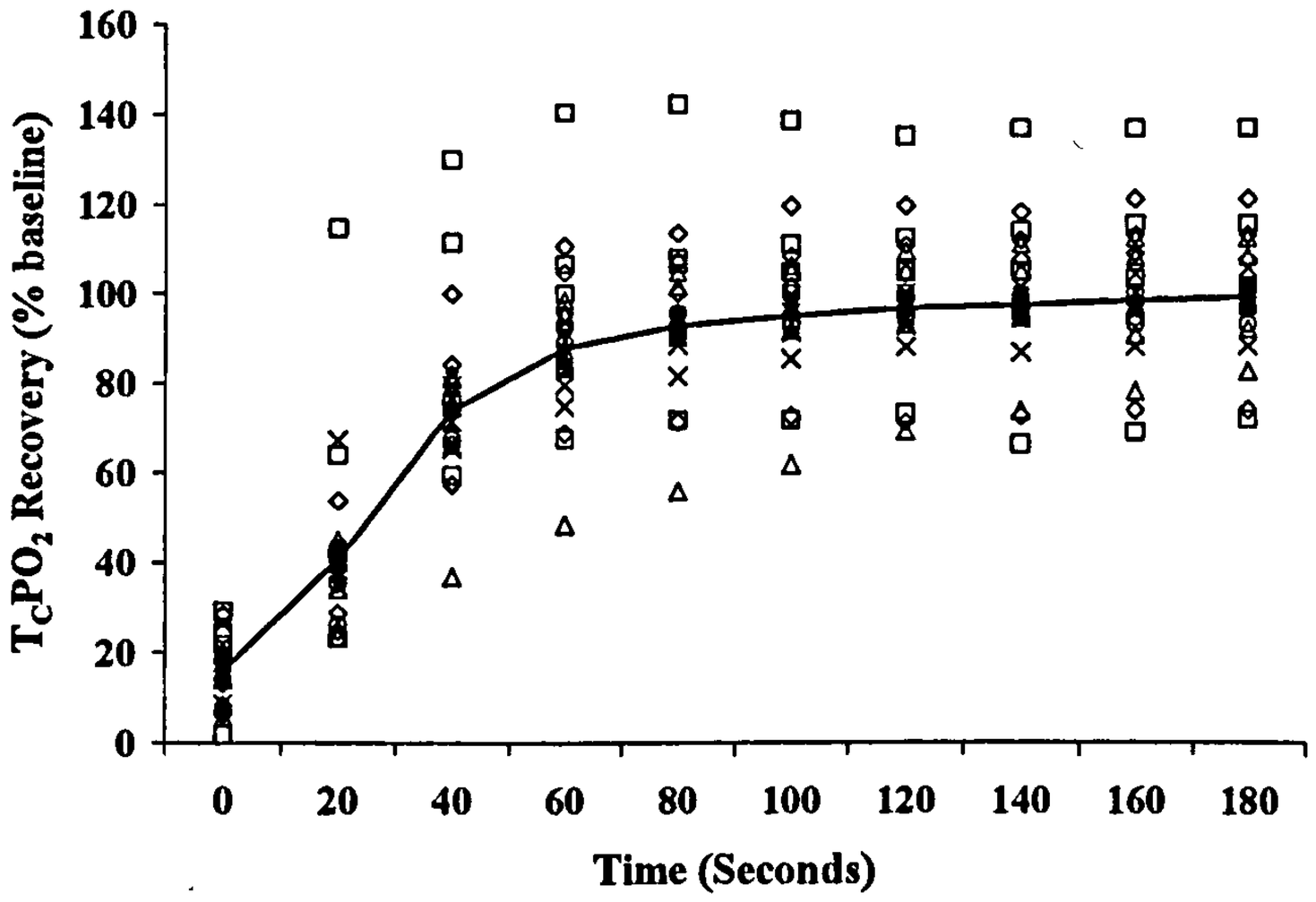

FIG. 5.3: RECOVERY OF TRANSCUTANEOUS OXYGEN PRESSURE (T_cPO_2) DURING 180 SECONDS FOLLOWING REMOVAL OF LOAD. AVERAGE DATA (CLOSED DIAMONDS AND SOLID LINE) AND INDIVIDUAL DATA FOR 5 PEOPLE (1 = OPEN DIAMONDS, 2 = OPEN SQUARES, 3 = OPEN CIRCLES, 4 = OPEN TRIANGLE AND 5 = CROSS).

Recovery data was based on n=3 for subject 3 only due to technical problems.

The time taken to reach 50 % baseline T_cPO_2 was 7-95 seconds. Intra-subject

CV ranged from 6.5-52.3 % (Table 5.4). Inter-subject CV ranged from 12.0- 85.1 % (Table 5.5).

Subject	$TcPO_2$ (50 % recovery)		
	Time (seconds)	SEM	CV
1	27.4	3.43	27.99
2	35.6	15.48	97.24
3	31.0	1.15	6.45
4	33.6	7.86	52.30
5	25.2	3.37	29.88
Mean	**30.6**	**6.26**	**42.77**

TABLE 5.4: TIME TAKEN FOR TRANSCUTANEOUS OXYGEN PRESSURE (T_CPO_2) TO REACH 50 % BASELINE T_CPO_2 AFTER THE REMOVAL OF LOAD FOR 5 PEOPLE ACROSS 5 TRIALS.

Trial	T_CPO_2 (50 % recovery)		
	Time (seconds)	SEM	CV
1	23.8	4.35	40.89
2	23.8	4.41	41.42
3	29.8	1.59	11.96
4	36.8	9.47	51.55
5	42.0	17.87	85.12
Mean	**31.2**	**7.54**	**46.19**

TABLE 5.5: TIME TAKEN FOR TRANSCUTANEOUS OXYGEN PRESSURE (T_CPO_2) TO REACH 50 % BASELINE T_CPO_2 AFTER THE REMOVAL OF LOAD FOR 5 TRIALS MEASURED ON 5 PEOPLE.

5.2.4.2 Experiment 2: T_CPO_2 recovery following the removal of a known mechanical load.

Average baseline and post-load T_CPO_2 (74.8 and 73.7 mmHg respectively) were slightly higher than experiment 1 but this did not attain statistical significance. The average, SEM and CV values for before and after load measurements are given in Table 5.6. CV for each subject across 5 trials ranged from 3.6-9.6 and 1.8-10.1 % at pre and post-load, respectively.

Subject	Pre-load T_CPO_2 (mmHg)			Post-load T_CPO_2 (mmHg)		
	Mean	SEM	CV	Mean	SEM	CV
1	79.2	1.36	3.83	79.6	1.50	4.22
2	62.6	2.69	9.62	62.0	2.81	10.14
3	72.6	1.78	5.48	71.4	2.50	7.84
4	81.2	1.32	3.63	77.0	0.63	1.84
5	78.6	2.42	6.89	78.6	3.12	8.89
Mean	**74.84**	**1.91**	**5.89**	**73.72**	**2.11**	**6.58**

TABLE 5.6: TRANSCUTANEOUS OXYGEN PRESSURE (T_CPO_2) PRE AND POST LOAD FOR 5 PEOPLE ACROSS 5 TRIALS.

Figure 5.4 shows recovery after the removal of load. Time taken to reach 50 % baseline values was 58-90 seconds. Intra-subject CV of time taken to reach 50 % baseline values ranged from 6.5-23.1 % (Table 5.7). Inter subject CV ranged from 23.3-41.7 % across the 5 trials (Table 5.8).

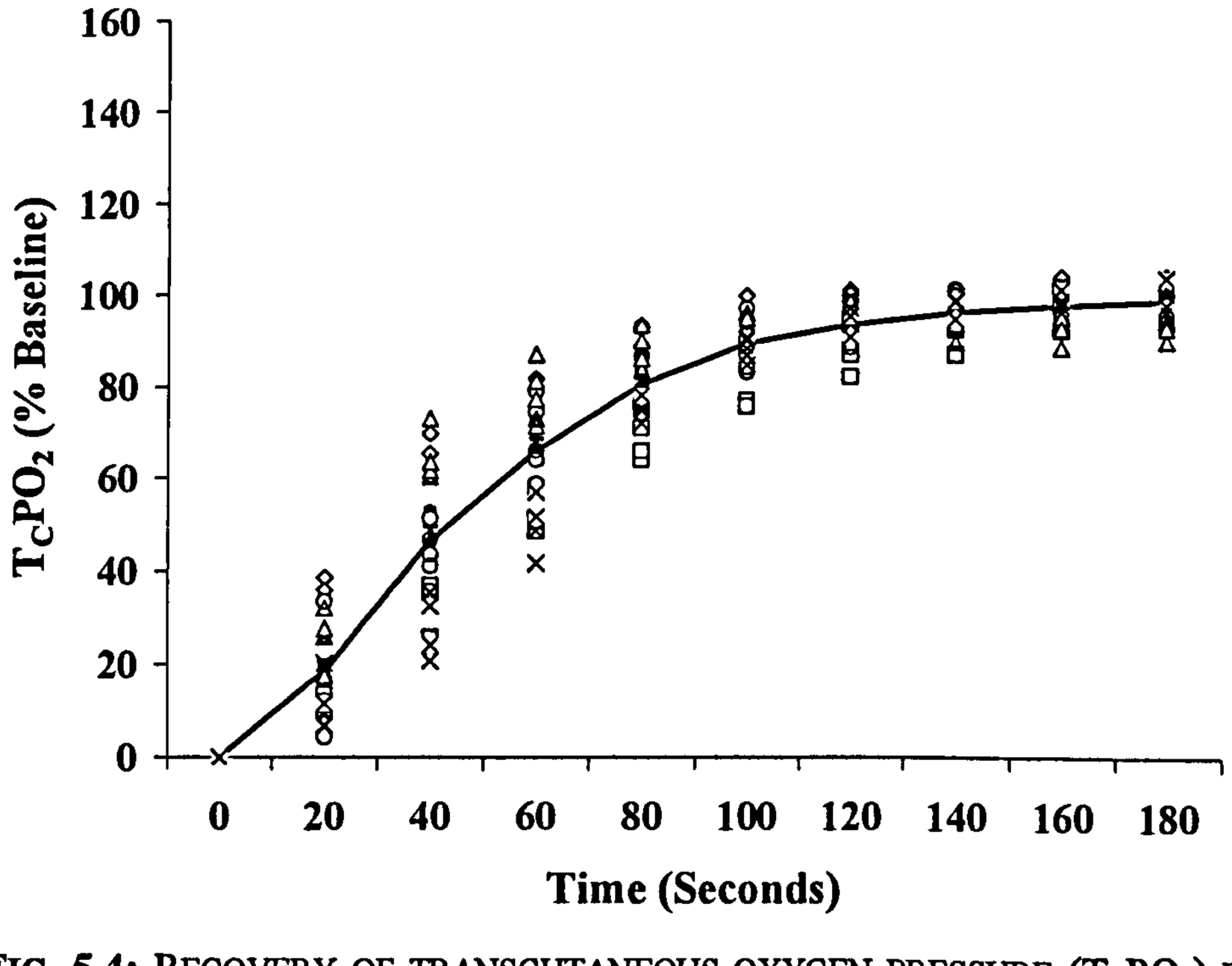

FIG. 5.4: RECOVERY OF TRANSCUTANEOUS OXYGEN PRESSURE (T_CPO_2) DURING 180 SECONDS FOLLOWING REMOVAL OF LOAD. AVERAGE DATA (CLOSED DIAMONDS AND SOLID LINE) AND INDIVIDUAL DATA FOR 5 PEOPLE (1 = OPEN DIAMONDS, 2 = OPEN SQUARES, 3 = OPEN CIRCLES, 4 = OPEN TRIANGLE AND 5 = CROSS).

Subject	T_CPO_2 (50 % recovery)		
	Time (seconds)	SEM	CV
1	31.6	2.06	14.61
2	54.8	4.21	17.19
3	44.6	1.91	9.59
4	28.6	2.96	23.14
5	59.6	1.72	6.45
Mean	**43.8**	**2.57**	**14.20**

TABLE 5.7: TIME TAKEN FOR T_CPO_2 TO REACH 50 % BASELINE VALUES AFTER THE REMOVAL OF LOAD FOR 5 PEOPLE ACROSS 5 TRIALS.

Trial	TcPO2 (50 % recovery)		
	Time (seconds)	SEM	CV
1	43.8	7.24	36.97
2	44.2	5.75	29.08
3	44.0	8.20	41.69
4	42.2	4.40	23.30
5	45.0	7.21	35.83
Mean	**43.8**	**6.56**	**33.37**

TABLE 5.8: TIME TAKEN FOR TRANSCUTANEOUS OXYGEN PRESSURE (T_CPO_2) TO REACH 50 % BASELINE T_CPO_2 AFTER THE REMOVAL OF LOAD FOR 5 TRIALS MEASURED ON 5 PEOPLE.

5.2.5 DISCUSSION

Baseline and post load T_CPO_2 values were not significantly different between the two experiments. Average CV of baseline values ranged from 3.8-10.6 (mean 7.4) and 3.6-9.6 (mean 5.9) % in experiments 1 and 2 respectively, which is similar to reported previously (Coleman et al., 1986). This variability was not due to inter-rater variation because the same tester carried out all experiments. Therefore it is likely that a large proportion of this variability was due to changes in blood pressure, local perfusion and tissue O_2 consumption. Local capillary distribution is also variable (Fig. 5.5, Chamberlin, 2006) and a direct correlation between T_CPO_2 and the density of superficial capillaries has been reported in people with chronic venous incompetence (Franzeck et al., 1984). Therefore

small changes in electrode position between trials could result in substantial variation in T_CPO_2 values. Three minutes after the removal of load T_CPO_2 was statistically similar to baseline values for both experiments 1 and 2. CV of post load T_CPO_2 ranged from 6.9-23.8 (mean 14.2) and 1.8-10.8 (mean 6.6) % for experiments 1 and 2 respectively, which was slightly higher than the variability in baseline values. Small changes in electrode contact with skin (due to the application and removal of load) and local oxygen pressure might cause these further variations after the removal of load.

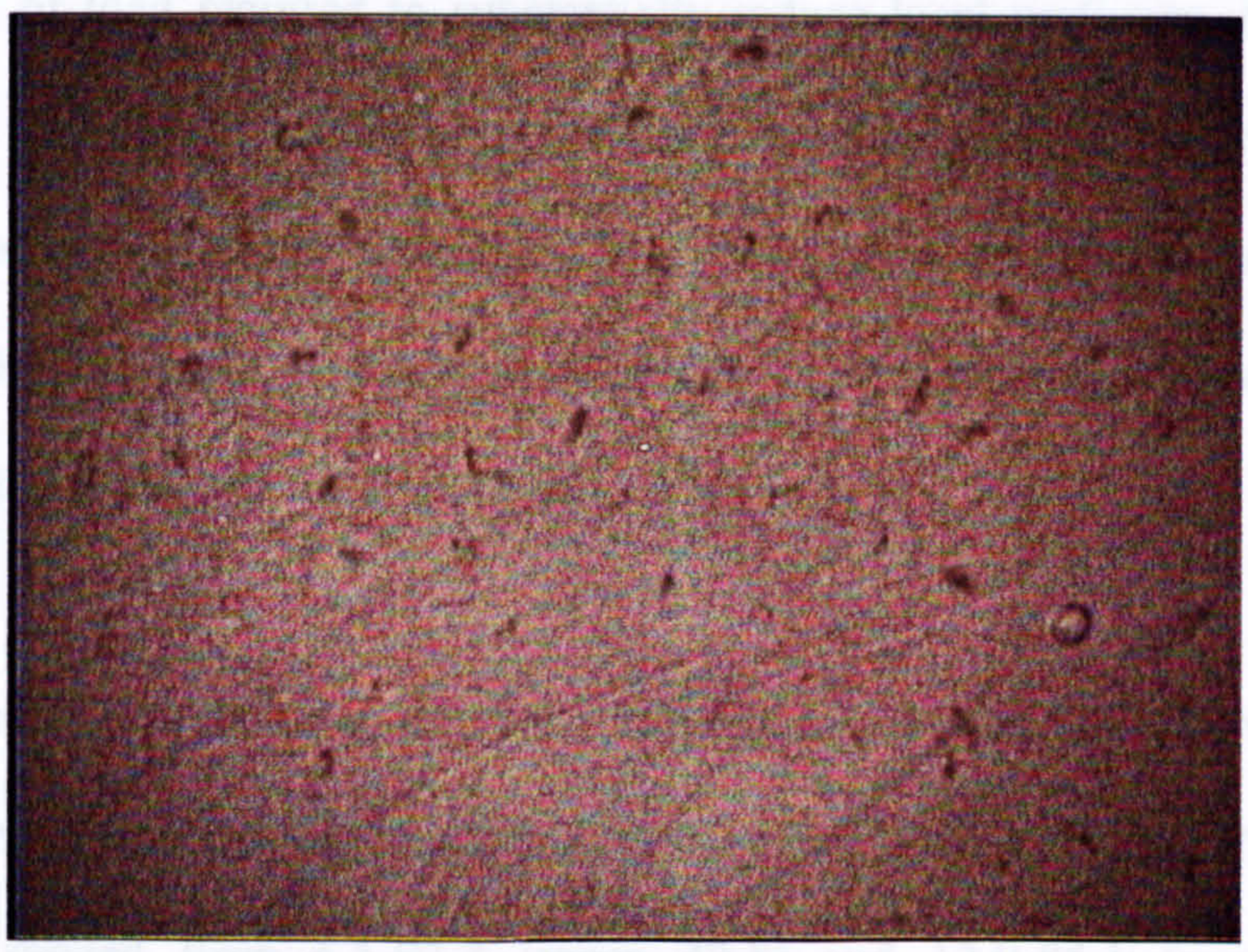

FIG. 5.5: IMAGE OF CAPPILLARY NETWORK AT THE SACRUM (1 MM^2) IN A PERSON WITH SPINAL CORD INJURY (CAPPILLARIES ARE THE SMALL DARK RED SPOTS IN THE IMAGE) (CHAMBERLIN, 2006).

5.2.5.1 Experiment 1: T_CPO_2 responses to mechanical loading and recovery.

Progressive loading resulted in gradual reductions in T_CPO_2 with some recovery after 5 minutes of load application. Similar patterns have been reported in response to progressive loading at the sacrum (Bader & Gant, 1985). However, across the 5 trials T_CPO_2 responses to given loads were highly variable (Fig. 5.2,

Table 5.3). Consequently the load required to cause T_CPO_2 to fall <20 mmHg was highly variable across trials for all 5 subjects (Table 5.2). Although Bader and Gant (1985) noted considerable inter-subject variations in the amount of tolerable load, such high intra-subject variability has not been reported previously. The pressure created at the sacrum by loads of 500, 700, 900, 1100, 1300 and 1500 g are 21.0, 29.4, 37.8, 46.2, 54.6 and 63.0 mmHg, respectively, and average systolic blood pressure is ~120 mmHg. It is therefore expected that a reduction to below 20 mmHg would occur at ≥1500 g in healthy AB subjects. It is possible that day to day variations in blood pressure significantly affected the amount of load required to substantially reduce local perfusion. In support of this, a non-linear relationship between baseline T_CPO_2 values and local arteriovenous pressure difference has been reported (Wyss et al., 1981). Alternatively, small changes in electrode placement might have resulted in substantial variations in tolerable load (Fig. 5.5).

Recovery during the three minutes following removal of load was variable (Fig. 5.3). The intra- and inter-subject CV for time taken to attain 50 % baseline T_CPO_2 ranged from 6.5-97.2 (mean 42.8) and 12.0-85.1 (mean 46.2) %, respectively. It is likely that these variations were due to differences in the amount and duration of load application resulting in varying levels of tissue hypoxia. Therefore, on some occasions reactive hyperaemia occurred resulting in very rapid recovery times whereas on others the mechanical stimulus was apparently too small to induce tissue hypoxia. This is confusing because, although the amount and duration of load application differed between trials, the

load was always high enough to reduce T_CPO_2 to < 20 mmHg - suggesting that the resulting tissue hypoxia should be similar.

The applied load increased progressively so that when higher loads were tolerated the total time of loading increased which might have resulted in the observed variations in recovery times. The amount that T_CPO_2 fell below 20 mmHg as a result of the final load might also have affected recovery times. Alternatively, differences in the load distribution at the sacrum (e.g. if the area at the sacrum was not completely flat) or daily variations in blood pressure and local perfusion pressure might have caused these variations. There is a day to day variation in blood pressure due to changes in heart rate and blood volume and viscosity.

5.2.5.2 Experiment 2: T_CPO_2 recovery following the removal of a known mechanical load.

As shown in Fig. 5.4, recovery during the 3 minutes following the removal of load was more consistent than during experiment 1. Intra- and inter-subject variability for time taken to recover to 50 % baseline values ranged from 9.6-23.1 (mean 14.2) and 23.3-41.7 (mean 33.4) %, respectively, which were smaller compared with experiment 1. The constant load and load duration was presumably the reason that the recovery profile was more consistent in this experiment. However, T_CPO_2 values (relative to baseline) were still variable until 60 seconds into recovery (Fig. 5.4). Similar to experiment 1, this might be due to variations in load distribution or blood pressure and local perfusion pressure. The applied load was apparently too small or applied for too short a duration to

induce reactive hyperaemia on the removal of load. Further work is required to identify a reliable experiment that does induce reactive hyperaemia. This would allow the assessment of capillary reactivity in people at risk of developing pressure sores and therefore their ability to recover from mechanical insults and tissue hypoxia.

5.2.6 CONCLUSIONS

Variability in baseline T_CPO_2 measurements was similar to that previously reported and is probably due to day-to-day variations in blood pressure, local perfusion and tissue O_2 consumption. T_CPO_2 responses under load and recovery from a load that reduces T_CPO_2 to <20 mmHg were highly variable. Recovery from a single known load was less variable than recovery from a load that reduces T_CPO_2 to <20 mmHg. It is therefore likely that baseline T_CPO_2 is not adequately reliable to distinguish differences in skin oxygenation between groups or in one group over time, in those not considered ischemic (40 mmHg). T_CPO_2 under load and recovery from a load that reduces T_CPO_2 to <20 mmHg is also unlikely to be adequately reliable. Recovery from a single known load might however be adequately reliable for this purpose.

5.3 AIMS

- To identify changes in resting T_CPO_2 and recovery times after a mechanical load in SCI people during a one year FES cycle training programme.

- To identify changes in peak seating pressures during a one year FES cycle training programme.
- To identify changes in muscle bulk and subcutaneous tissue surrounding the ischeum during a one year FES cycle training programme.

5.4 METHODOLOGY

Five SCI people (described previously, see Chapter 2) were tested for T_CPO_2 responses to progressive loading and time taken for recovery from a set applied load at three monthly intervals through out a one year FES cycling training programme. This aimed to assess any changes in the load required to reduce T_CPO_2 <20 mmHg and recovery from load due to the training programme.

The same people were also tested for peak seating pressures in i) a standard (NHS) and ii) their own wheelchair at three monthly intervals and for gluteal muscle bulk and muscle and subcutaneous tissue thickness over the ischeum before and after the training programme. This aimed to assess whether FES cycling improved gluteal muscle bulk and whether this resulted in alterations in peak seating pressures during wheelchair sitting.

5.4.1 TRANSCUTANEOUS OXYGEN PRESSURE (T_CPO_2)

T_CPO_2 measurements were used to assess the response of the microcirculation to metabolic demands of the surrounding tissue due to application of load. Measurements were made at the sacrum because the technique must be carried out at a flat area of skin. Also it is clinically significant to take these

measurements at the sacrum because SCI people are particularly vulnerable to pressure sores in this area.

The experimental system (Bader and Gant, 1985, 1988) was set up at described previously (Fig. 5.1). A period of 15 minutes was given to allow equilibrium to be attained, and the baseline T_CPO_2 value was recorded. A load of 3000 g was then applied to the loading pan and left for three minutes and the lowest T_CPO_2 value was recorded. Load was then removed and T_CPO_2 level was recorded every 20 seconds for three minutes. Time taken to 25, 50 and 75 % of baseline T_CPO_2 values were calculated for each subject at each time point.

5.4.2 SEATING PRESSURE

Seating pressures were measured using a force sensing array system (Force Sensitive Applications, Canada) that provides direct information on the pressure created at the body-supporting surface interface at a frequency of 10 Hz. The system uses a square mat approximately 500 x 500 mm consisting of an array of 20 x 20 25 mm^2 sensors capable of capturing pressure data in real time. Visual outputs are provided on a computer screen. The pressure mat was calibrated according to the manufacturer's guidelines.

At each time point two tests were carried out, first with the subject seated on a standard NHS wheelchair and cushion (depth 8.0 cm) and second with their own personal wheelchair and cushion. The test consisted of 10 scans each taken 30 seconds after a 2 second pressure lift in the chair. For each scan the subject was in a standard sitting position with the tops of the thighs horizontal to the floor

(verified by a spirit level) by adjustment of footplate height, feet flat on footplates, hands resting in lap and looking directly forwards at a fixed point on the wall (Fig. 5.6). The maximum pressure under each ischial tuberosity was taken and averaged over the ten measurements.

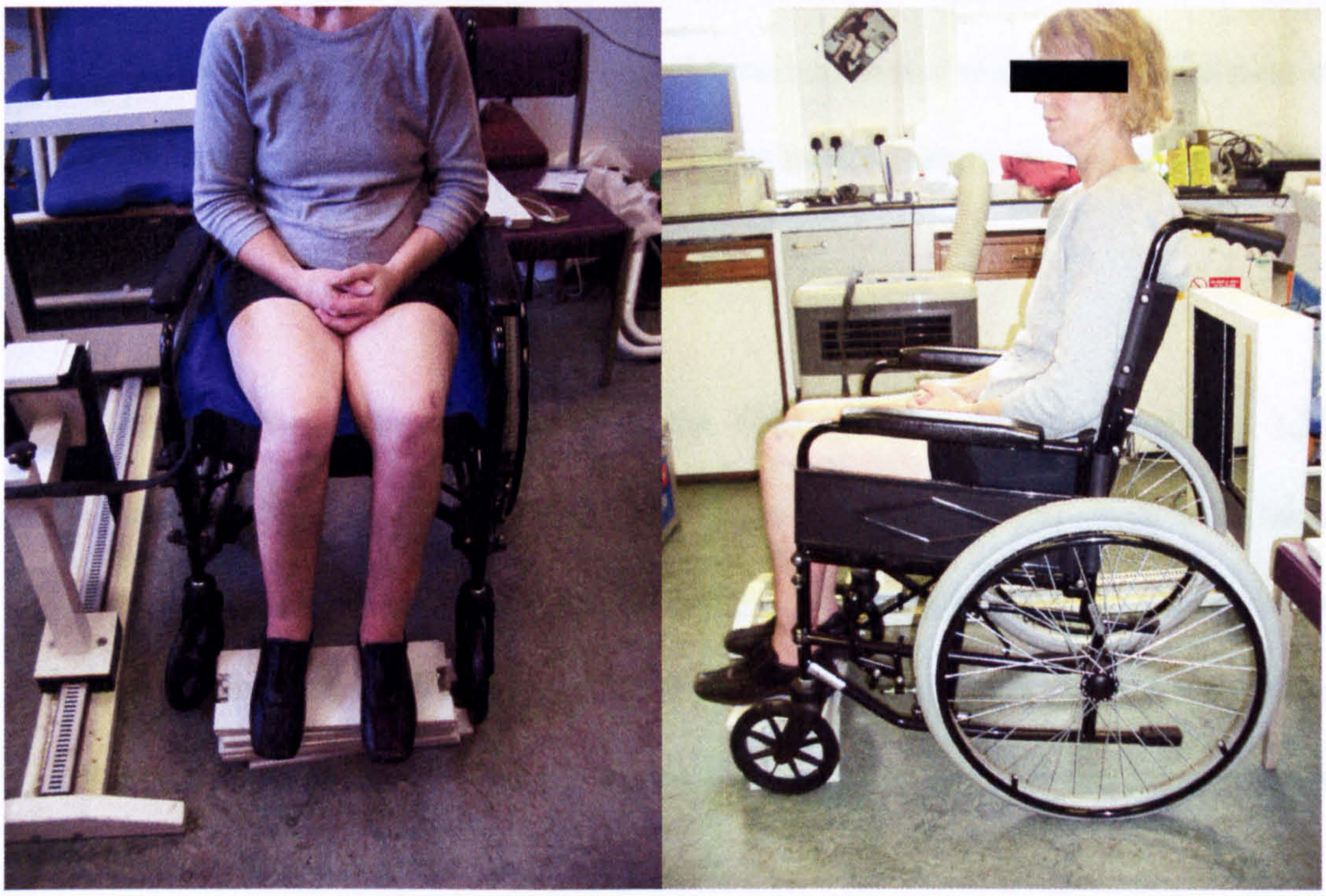

FIG. 5.6: SEATING PRESSURE MEASUREMENTS TAKEN IN A STANDARD WHEELCHAIR. FEET ARE RAISED SO THAT THIGHS ARE HORIZONTAL AND ARMS ARE RESTING IN LAP.

Despite regular calibration of the pressure mat at 6 month intervals as recommended by the manufacturer, mean and peak pressures appeared to drift over time resulting in values substantially lower than previously reported after three months. Since it has been reported that no changes in mean overall pressure occur as a result of an ES training programme (Bogie & Triolo, 2003), i.e. body mass remains constant, 3-12 month data was corrected so that the same overall

pressure was applied for each subject at all time points and the resultant peak pressures were recorded.

5.4.3 GLUTEAL THICKNESS

At baseline and after 12 months FES cycle training magnetic resonance imaging (MRI) was used to assess gluteal muscle cross sectional area and the thickness of muscle and subcutaneous tissue between the ischeal tuberosity and the surface of the skin.

Subjects were positioned prone in the MRI scanner (Phillips, Acheiva 1.5T (Release 1.5) using the body coil). A series of coronal (frontal) slices were taken through the pelvis to find the lowest point of the ischia. A line was then drawn between these and a slice was taken of this transverse section. Images were 8mm thick, 2mm spacing and taken without a secondary coil as this caused some distortion of the tissues.

Cross-sectional area of the gluteals were measured using custom designed software (Matlab programming; Woledge, 2006, unpublished). This software was also used to measure muscle and subcutaneous tissue thickness over the ischia. A line was drawn between the two most posterior points of the ischia. Two lines were the drawn perpendicular to this from each ischia to the surface of the skin and the distance of muscle and subcutaneous tissue were recorded.

5.4.4 DATA ANALYSIS

Analysis of variance was carried out on all T_CPO_2 and seating pressure data throughout the one year training programme. Paired Students T-Tests were used to assess changes in gluteal muscle size and muscle and subcutaneous tissue thickness covering the ischia.

5.5 RESULTS

5.5.1 TRANSCUTANEOUS OXYGEN PRESSURE (T_CPO_2)

Data could not be collected on one SCI person due to deformality at the sacrum from a previous pressure sore, therefore all T_CPO_2 data was based on n=4 except baseline which was based on n=3 due to technical problems with one person. Average baseline T_CPO_2 at baseline, 3, 6, 9 and 12 months were 68.5, 70.8, 66.3, 60.3 and 63.8 mmHg, respectively. After removal of load and a three minute recovery period, values were similar to baseline ($P > 0.05$, Fig. 5.7). Both pre- and post-load T_CPO_2 values did not change significantly throughout the one year training programme.

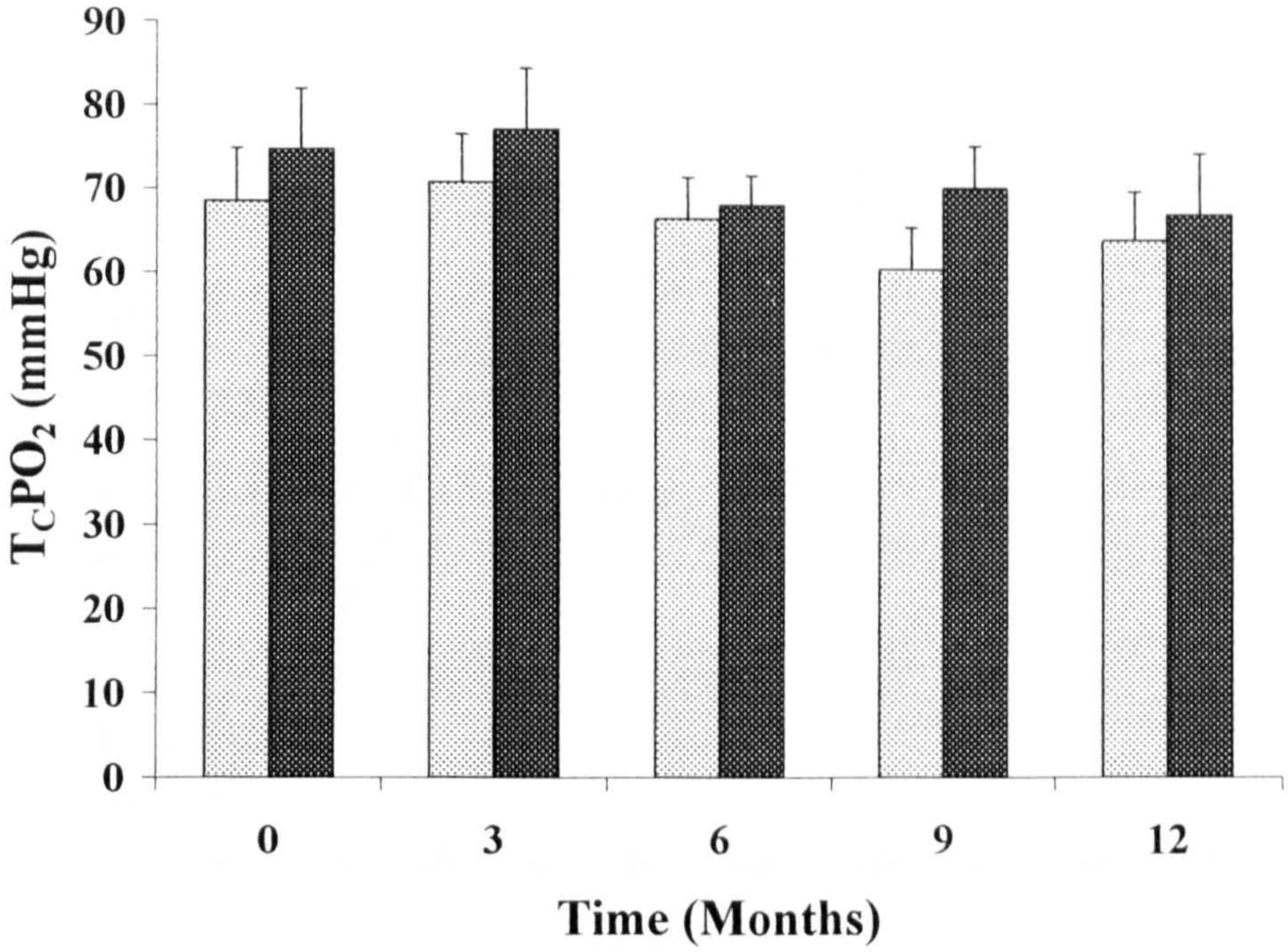

FIG 5.7: TRANSCUTANEOUS OXYGEN PRESSURE AT BASELINE (GREY BARS) AND 3 MINUTES AFTER THE REMOVAL OF LOAD (BLACK BARS) FOR 5 **SCI** PEOPLE THROUGH OUT THE ONE YEAR TRAINING PROGRAMME.

Average recovery after the removal of load at baseline, 3, 6, 9 and 12 months is shown in Fig. 5.8. Recovery rate did not show any substantial change as a result of the training and time taken to reach 25, 50 and 75 % baseline T_CPO_2 did not change significantly through out the one year training programme ($P > 0.05$, Table 5.9).

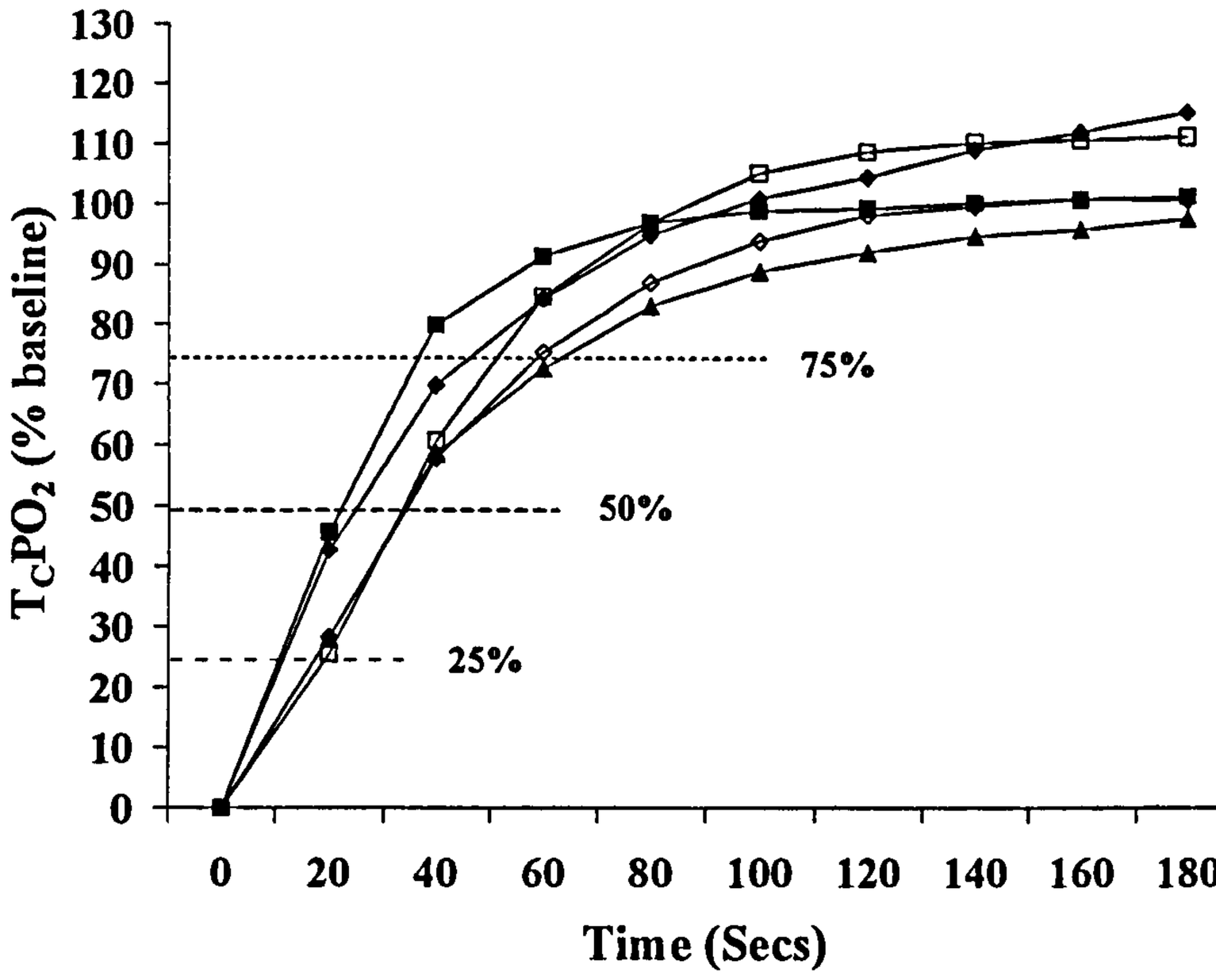

FIG. 5.8: AVERAGE RECOVERY OF TRANSCUTANEOUS OXYGEN PRESSURE (T_cPO_2) DURING 180 SECONDS FOLLOWING REMOVAL OF LOAD FOR 5 SCI PEOPLE THROUGH OUT THE ONE YEAR TRAINING PROGRAMME (BASELINE = CLOSED DIAMONDS, 3 MONTH = CLOSED SQUARES, 6 MONTH = CLOSED TRIANGLES, 9 MONTH = OPEN SQURES AND 12 MONTH = OPEN DIAMONDS).

% Recovery	Time (Seconds)				
	Baseline	3 Month	6 Month	9 Month	12 Month
25	17.7 (6.4)	18.3 (5.5)	19.3 (4.5)	19.5 (2.2)	23.0 (8.7)
50	32.7 (11.6)	29.0 (7.8)	35.3 (6.5)	33.5 (2.1)	37.3 (10.3)
75	53.3 (18.4)	42.3 (12.1)	59.0 (9.0)	52.0 (3.2)	53.8 (11.6)

TABLE 5.9: TIME TAKEN (SEM) TO ATTAIN 25, 50 AND 75 % BASELINE T_cPO_2 AFTER THE REMOVAL OF LOAD THROUGH OUT THE ONE YEAR TRAINING PROGRAMME.

5.5.2 SEATING PRESSURE

An example of a seating pressure reading taken at baseline is shown in Fig. 5.9. Left and right peak seating pressures measured in a standard wheelchair and cushion did not change significantly throughout the training programme ($P > 0.05$, Fig. 5.10). Peak pressures measured in each subject's own wheelchair also

did not change significantly (P >0.05, Fig. 5.11). Peak pressures measured in each subject's own wheelchair were generally lower than those measured in an NHS wheelchair, but this was not statistically significant.

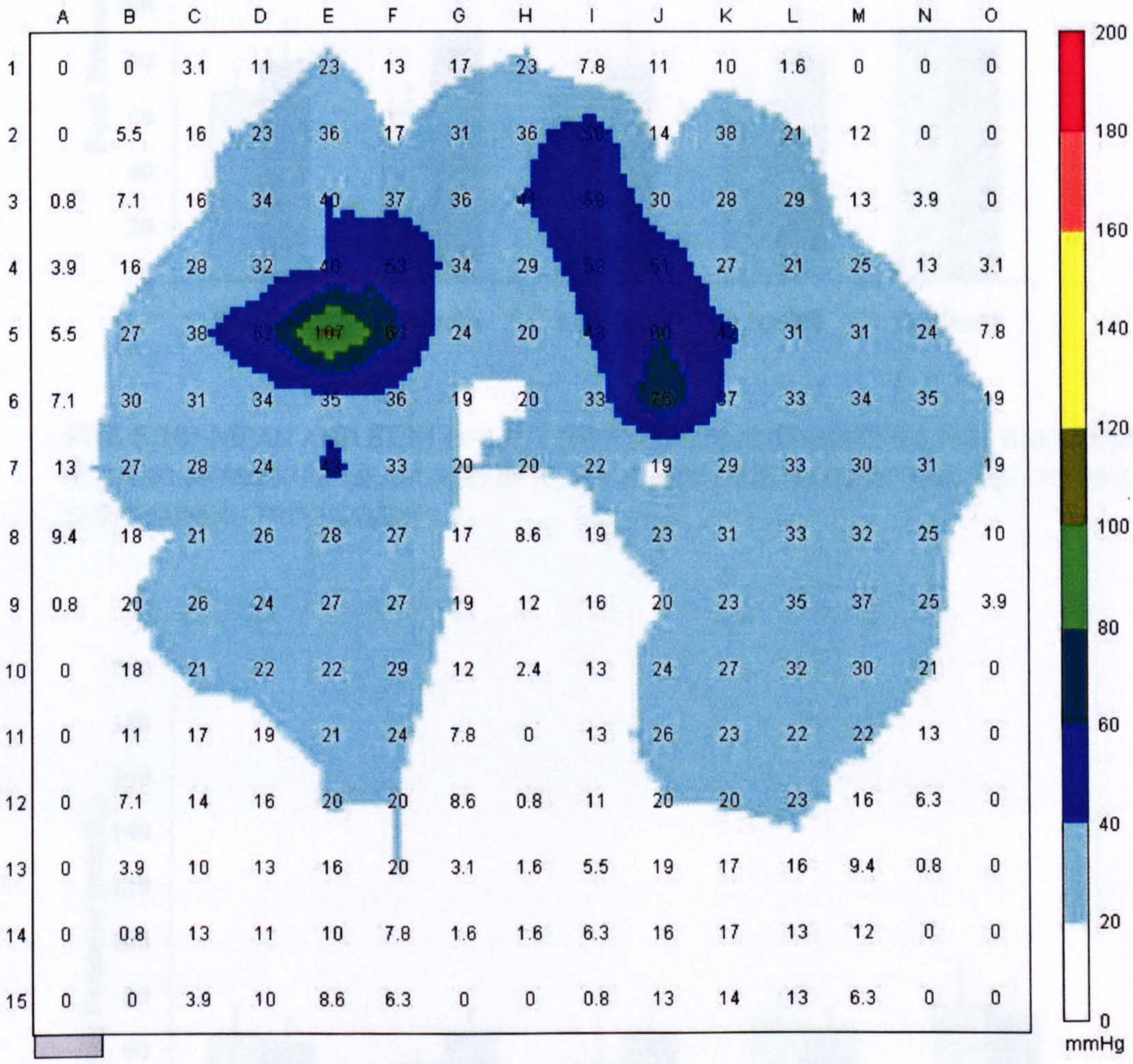

FIG. 5.9: TYPICAL SEATING PRESSURE SCAN TAKEN FROM ONE SCI PERSON AT BASELINE.

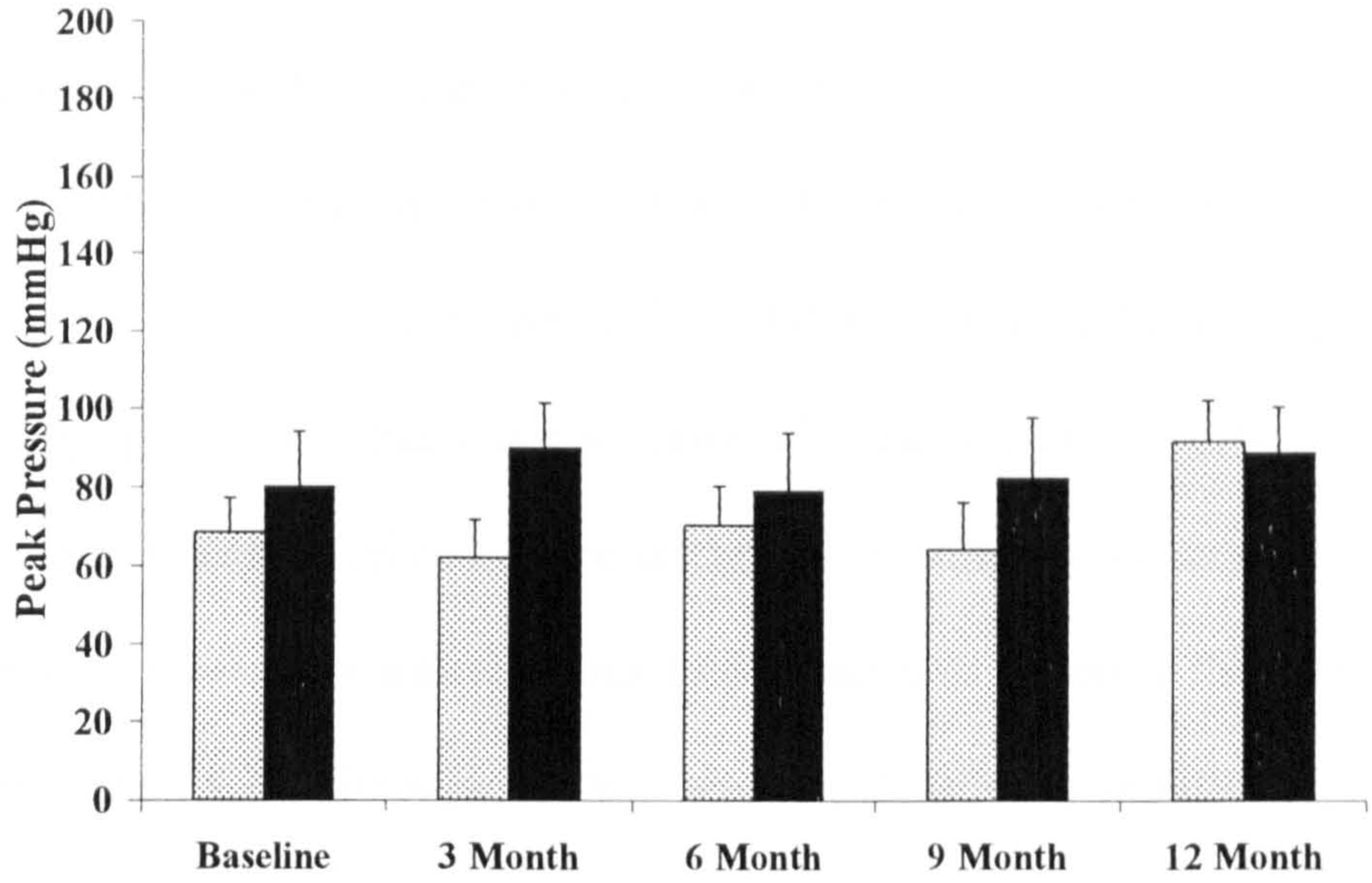

FIG. 5.10: MEAN AND **SEM** OF LEFT (GREY BARS) AND RIGHT (BLACK BARS) PEAK SEATING PRESSURES MEASURED IN A STANDARD **NHS** WHEELCHAIR THROUGHOUT THE TRAINING PROGRAMME.

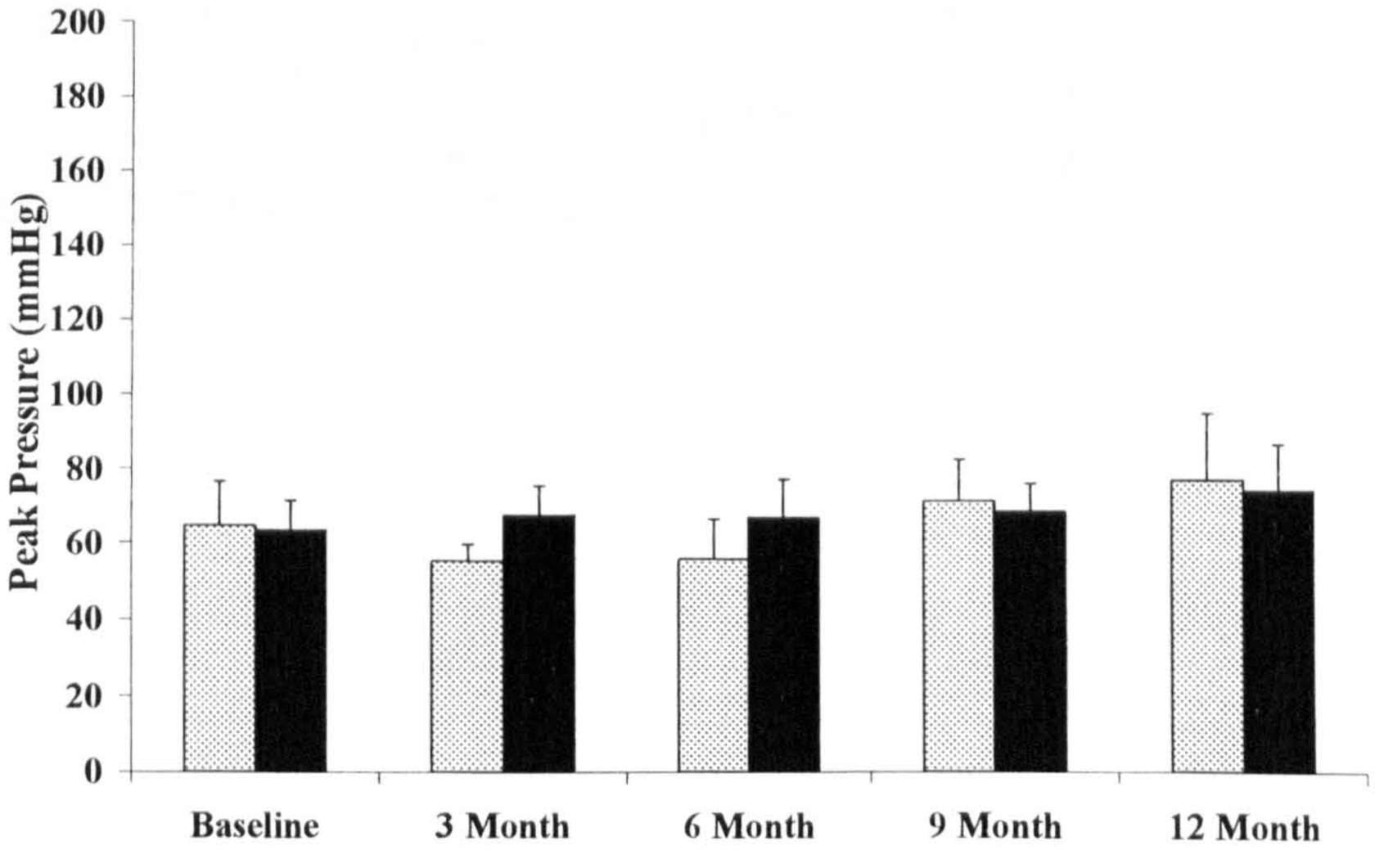

FIG. 5.11: MEAN AND **SEM** OF LEFT (GREY BARS) AND RIGHT (BLACK BARS) PEAK SEATING PRESSURES MEASURED IN EACH SUBJECT'S OWN WHEELCHAIR THROUGHOUT THE TRAINING PROGRAMME.

5.5.3 GLUTEAL THICKNESS

An example of the MRI scans taken at baseline and 12 months of the transverse section at the lowest point of the ischia is shown in Fig. 5.12. CV for repeated measures of the gluteal area was 2.2 %. Gluteal area tended to improve with training (Fig. 5.13) but this was not statistically significant (P = 0.10 and 0.09 for right and left, respectively). Muscle thickness covering the ischia did not change after training, however subcutaneous tissue thickness covering the ischia tended to reduce (Fig. 5.14) although this was also not statistically significant.

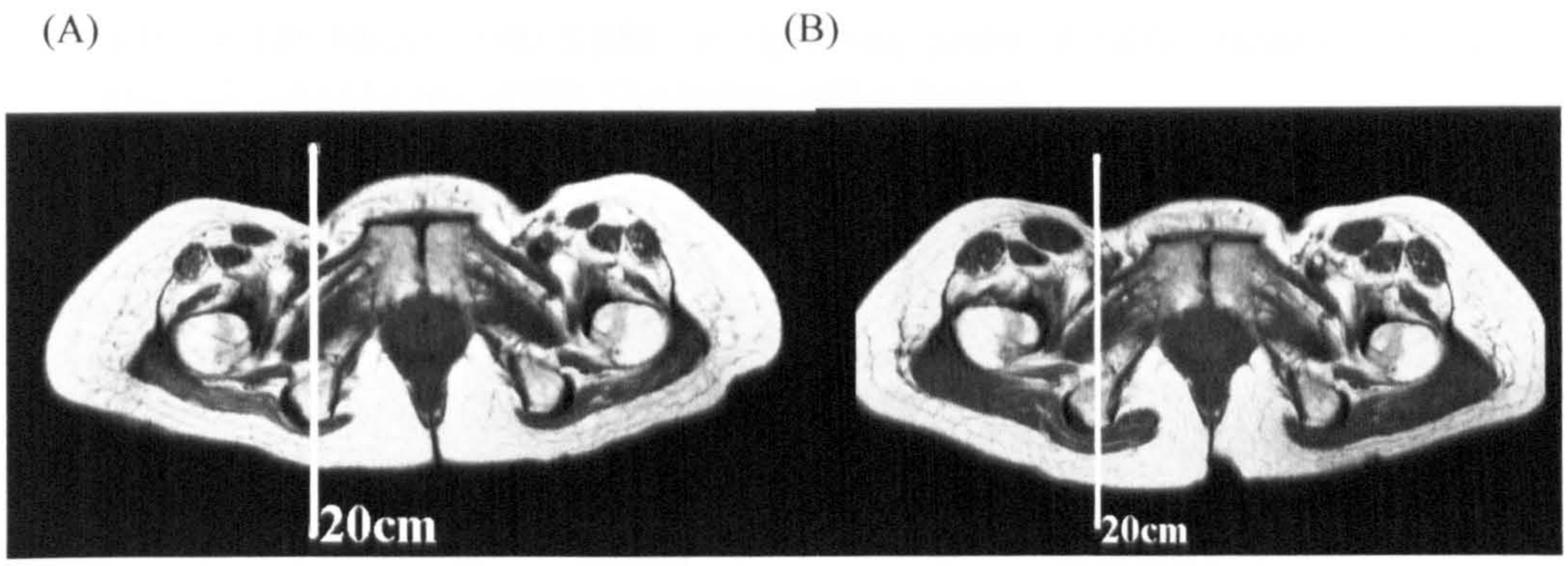

FIG. 5.12: TYPICAL MRI IMAGE OF A TRANSVERSE SECTION THROUGH THE LOWEST POINT OF THE ISCHEALS AT (A) BASELINE AND (B) AFTER 12 MONTHS TRAINING IN ONE SCI SUBJECT.

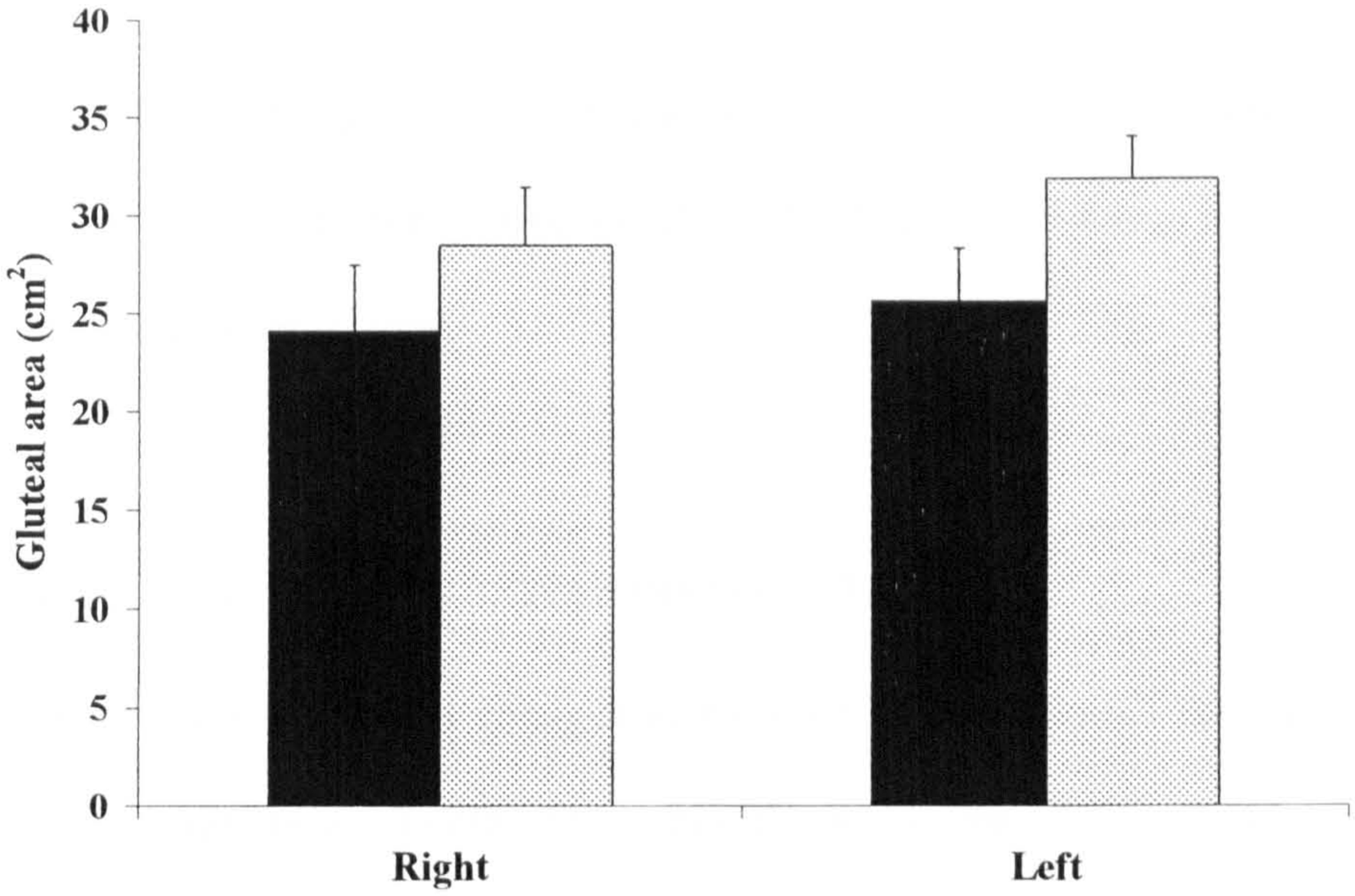

FIG. 5.13: MEAN AND **SEM** OF GLUTEAL MUSCLE SIZE MEASUED AT BASELINE (BLACK BARS) AND AFTER TRAINING (GREY BARS).

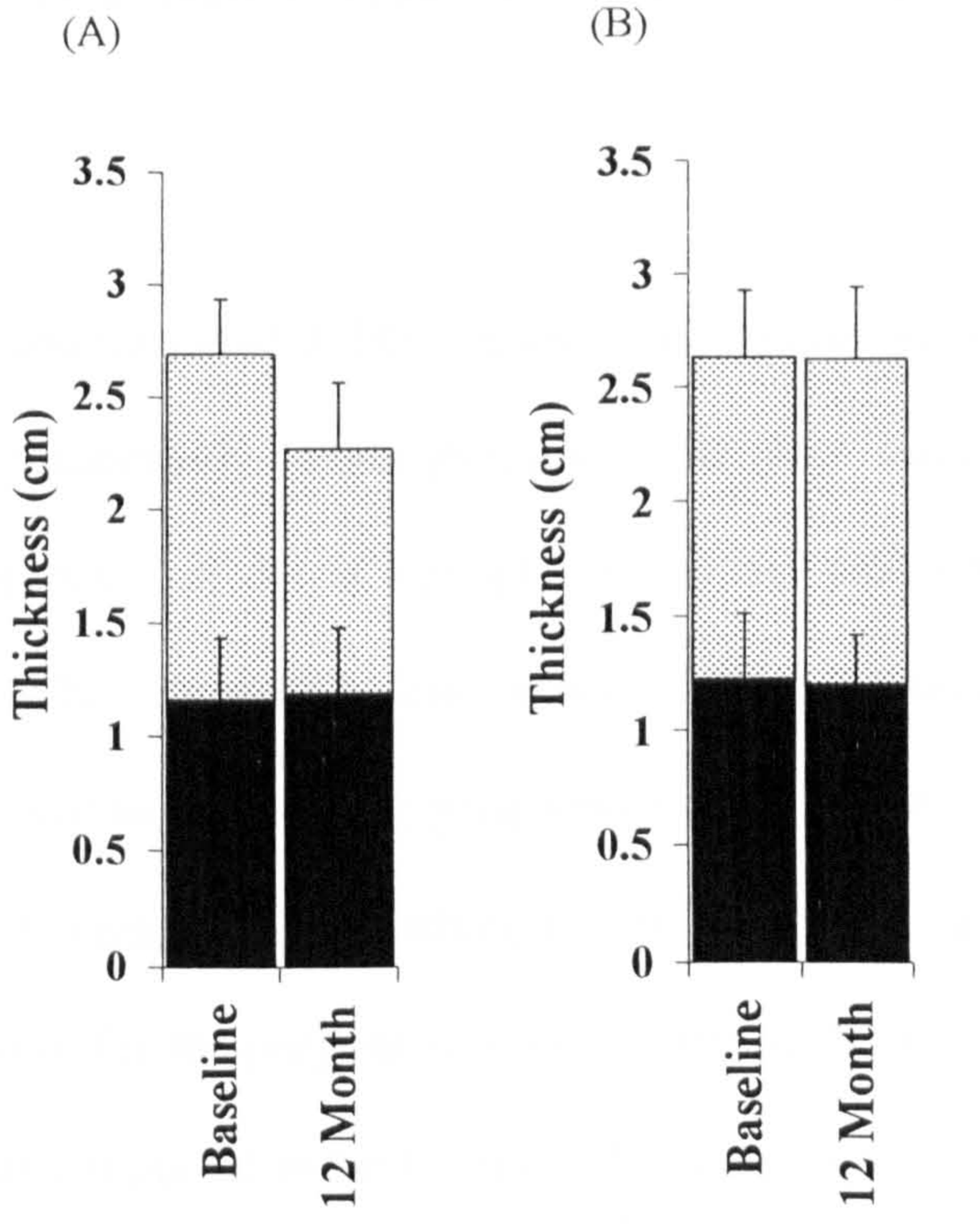

FIG. 5.14: MEAN AND **SEM** OF MUSCLE (BLACK BARS) AND SUBCUTANEOUS TISSUE (GREY BARS) THICKNESS COVERING THE RIGHT (A) AND LEFT (B) ISCHEALS AT BASELINE AND AFTER TRAINING.

5.6 DISCUSSION

No significant changes were found in any of the variables measured although there was a tendency for gluteal muscle and subcutaneous tissue size to increase and decrease, respectively.

5.6.1 TRANSCUTANEOUS OXYGEN PRESSURE (T_cPO_2)

There were no significant changes in baseline T_cPO_2 through out the one year training programme. T_cPO_2 following three minutes recovery also did not change significantly as a result of the training programme (Fig. 5.7). There were also no significant changes in the recovery profile following the removal of load (Fig. 5.8) and the time taken to attain 25, 50 and 75 % baseline T_cPO_2 values (Table 5.9).

Variability in pre and post load T_cPO_2 values in AB people reported here were 6-7 and 7-14 %, respectively. Throughout the one year training programme, average intra subject CV of the SCI people for pre and post load T_cPO_2 values were 14.5 and 11.4 %, respectively. This indicates that any changes which might have taken place due to the training programme were less than the day to day variability in the measurement technique and thus the technique is not sufficiently sensitive for the purpose required in this study. This is in agreement with data previously reported in hemiplegics (Diamantopoulos et al., 1995). Fig. 5.5 indicates the variability in the capillary network at the sacrum of an SCI person (Chamberlin, 2006). Small changes in electrode placement over the year might therefore have contributed to this variability.

It is possible that the recovery profile occurred because the applied load was not large enough or was applied for a too small duration to induce a reactive hyperaemic response. The recovery profile in SCI people was similar to that of AB people in response to a similar load (Fig. 5.4 and 5.8 for AB and SCI people, respectively) and therefore is probably not due to impairments in vessel reactivity due to adaptations caused by SCI. The recovery profile and time taken to attain 25, 50 and 75 % recovery did not change as a result of the training indicating that vessel reactivity did not improve due to a one year training programme. It is possible that changes did not occur because the applied load was insufficient to induce a hyperaemic response. In agreement loads of 200-2500g have been shown to be insufficient to induce reactive hyperaemia in both AB and SCI people. This suggests that the load was not great enough to induce tissue hypoxia, and thus it would not be surprising that there was no change in the recovery time. Further research is required to define a load and load duration that causes sufficient tissue hypoxia and therefore reactive hyperaemia. This test could then be used to assess whether vessel reactivity improves as a result of training, given that the test was reasonable for the purpose of the study. It should be noted that this test can be time consuming and uncomfortable for SCI people. It should therefore be ensured that the sensitivity of the test be adequate to highlight potential changes. It is possible that the use of alternative equipment (e.g. Laser Doppler) would be more appropriate for this purpose.

5.6.2 SEATING PRESSURE AND GLUTEAL THICKNESS

Peak seating pressures measured in a standard (NHS) and each subject's own wheelchair did not change as a result of an intense long term programme of FES

cycling (Fig. 5.10 and 5.11, respectively). Gluteal muscle size tended to improve with training (Fig. 5.12 and 5.13), but this was not statistically significant and there was no concomitant change in muscle thickness covering the ischeal tuberosities (Fig. 5.14). Subcutaneous tissue covering the ischia tended to reduce after training (Fig. 5.14) but this was not significant.

It is likely that the small change in muscle bulk and slight reduction in subcutaneous tissue covering the ischia resulted in no changes in peak seating pressures after training. This is in disagreement with a previous study that showed significant reductions in mean ischeal region pressures after an 8 week programme of FES standing (Bogie & Triolo, 2003). Garber & Krouskop (1982) reported that higher pressures over bony prominences occurred in thin rather than average weight or obese people. Therefore it is possible that no changes in peak seating pressures occurred in the present study because of the variability in body size of the 5 subjects (approximate body mass index range = 19-28, see Chapter 2).

It should also be noted that the analysis used in the present study for peak seating pressures was limited because it was assumed that overall pressure for each subject did not change throughout the training programme due to problems with pressure mat calibration. It is possible that this assumption was incorrect because gluteal muscle size did tend to change after training, which would affect overall pressures, although these changes were not significant. It has also been reported that that the FSA is inaccurate with repeated measures varying by –4.5 % to +27.3 % for the model used in the present study measured at human body

temperature (Fergus et al., 2003). The study also found the recorded average pressure to increase by up to 21 % over a 30 minute period. It is likely that these inaccuracies caused the drift seen in measurements over time in the present study. The peak seating pressure results in the present study should therefore be interpreted with caution, and this might explain the discrepancy between studies.

The fact that gluteal muscle cross sectional area did tend to improve with training (Fig. 5.13) indicates that pressure distribution at the body-support interface did change due to the training programme. Therefore peak seating pressures were potentially reduced in some SCI people. However since there was a concomitant reduction in subcutaneous tissue thickness covering the ischia in some SCI people, the positive effects of increased muscle bulk might have been reduced.

5.7 Conclusions

Baseline T_cPO_2, T_cPO_2 under load and recovery time from the removal of load did not change throughout the training programme. This indicates that improvements in cutaneous tissue oxygenation and vessel reactivity did not improve due to the training. However, the T_cPO_2 reliability study identified large day to day variations in T_cPO_2 values suggesting that the test was not discriminative enough to identify potential changes due to the training programme. Furthermore the recovery from load profile showed that reactive hyperaemia did not occur and this was similar in SCI and AB people. It should be noted that this methodology is time consuming and uncomfortable for SCI people. Therefore a reasonable and discriminative test should be designed if this methodology were to be reused.

Peak seating pressures, gluteal muscle size and muscle and subcutaneous tissue thickness covering the ischia did not change due to the training. However gluteal muscle size did tend to improve and subcutaneous tissue covering the ischia tended to reduce. It is therefore likely that these small changes resulted in no changes in peak seating pressures. However seating pressure data should be taken with caution due to limitations with calibration.

Overall, these results suggest that an intense long term FES cycling programme was inadequate to reduce pressure sore susceptibility in SCI people. However, limitations in both the tissue oxygenation and seating pressure methodologies might have affected these results and therefore should be interpreted with caution.

The following chapter considers power output and the metabolic responses during 10 minutes cycling at 100 % stimulation intensity and during a one hour training session simulation in SCI people after the one year training programme.

Chapter 6 Control of cardiopulmonary responses and substrate metabolism in exercise - The effect of FES cycling

Following the one year training programme power output remained low in the 5 SCI people. It was additionally observed during home training sessions and exercise tests that rapid fatigue occurred during the initial few minutes of exercise and subsequently two partial recovery periods were apparent. The aim of this chapter was to investigate the metabolic responses during FES cycling and how they relate to the changes in power output during FES cycling. It was hoped that this knowledge would contribute to the understanding of the relatively low power outputs attained during FES cycling and how current FES protocols can be altered in terms of stimulation parameters to optimise performance.

Metabolic responses were measured in the 5 SCI people after one year of FES cycle training during i) 10 minutes FES cycling at 100 % stimulation intensity in order to assess peak power output and fatigability during FES cycling and ii) a one hour FES cycling training session (as described in Chapter 2) to assess power output and metabolic responses during home training sessions.

Power output and related metabolic responses during FES cycling in SCI people are important because, despite a long term intense programme of FES cycling, power output remained generally too low for outdoor recreational cycling and insufficient

to induce substantial health benefits as would be expected following such an intense exercise regimen in AB people.

6.1 Literature Review

Both central command (feed-forward) and neural reflex (feedback) mechanisms play an important role in the control of cardiovascular, respiratory and metabolic responses to voluntary exercise. The central command theory (Krough & Lindhard, 1913) proposes that a drive to exercise is initiated by the cerebral cortex to recruit motor units and initiate the cardiovascular responses. Alternatively the feedback mechanism proposes that the muscular contractions stimulate afferent nerve fibres to mediate a reflex response. Substantial evidence also exists that blood-bourn metabolic stimuli bring about the cardiovascular and substrate metabolism responses. Much work has been carried out in an attempt to elucidate the relative contribution of each mechanism to exercise responses. Experiments have been performed involving exclusion or partial abolition of central command (electrical stimulation and neuromuscular blockade respectively), abolition of neural reflexes (sensory blockade or anaesthesia) and abolition of both mechanisms (spinal cord injury).

6.1.1 Cardiovascular and respiratory responses to exercise:

During exercise in healthy AB humans, increases in heart rate (HR), cardiac output (Q) and ventilation (V) occur to increase the supply of oxygen (O_2) to working muscles. In turn, oxygen uptake ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) are

increased. Significant increases in HR, Q, V and $\dot{V}O_2$ have been shown to occur in the absence of central command i.e. during ES, in animals (Kao & Ray, 1954) and humans (Adams et al., 1984a; Strange et al., 1993). HR and ventilatory responses to exercise have also been shown to be unaffected by the removal of afferent reflex responses (epidural anaesthesia) in AB people (Fernandes et al., 1990).

It has been suggested that stimulation of the arterial chemoreceptors is a major determinant in the non-linear increase in V at higher work rates since this response is abolished in patients without carotid bodies (Wasserman et al., 1975). The possibility that this response is mediated by acidosis has been disputed since patients who cannot catabolise glycogen and create lactic acid during exercise (McArdles Syndrome) show a similar response (Patterson et al., 1990). Although no direct evidence exists there is strong support for the theory that the rise in arterial plasma potassium (K^+) concentration (hyperkalaemia), that occurs due to a rapid K^+ loss from the working muscles at the onset of exercise, stimulates the arterial chemoreceptors (Patterson, 1992). A close temporal relationship between arterial [K^+] and V during exercise has been identified in both healthy subjects and patients with McArdles Syndrome (Patterson et al., 1990). Animal studies by Paterson and colleagues (1992) have shown that hyperkalaemia increased ventilation up to 40 and 250% during normoxia and hypoxia respectively. It was concluded that a combination of hyperkalaemia and hypoxia generates a powerful drive to breathing (Paterson et al., 1992).

Exercise-induced increases in SV in the absence of feed-forward and feedback mechanisms is possibly due to improved venous return to the heart induced by muscular contraction (Frank-Starling mechanism). It has been suggested that humoral feedback is of importance for the HR response to exercise (Kjær et al., 1999). A rise in HR has been demonstrated during ES induced leg exercise in SCI people (Brown et al., 1990; Kjær et al., 1999) indicating that a blood bourn stimulus mediates the HR response to exercise (Raymond et al., 2000). Catecholamines secreted by the adrenal medulla in response to stress bring about increases in HR in AB persons. Bloomfield et al. (1994) identified a significant catecholamine response to ES exercise in SCI people, albeit substantially less than that of AB people exercising voluntarily. Since innervation of the adrenal medulla derives from levels T5-T9 the catecholamine response to exercise might be impaired in SCI people with lesions above T9 resulting in a reduced HR response. A circulatory hypokinetic response (lower Q for a given $\dot{V}O_2$) has also been noted in SCI people that could be the consequence of a lower HR response to a given work rate due to an inefficient rise in catecholamines or to the inactivity of the venous muscle pump and consequent venous pooling in the lower limbs (Jacobs et al., 2002).

Increases in mean arterial blood pressure have also been shown to occur in the absence of central command (Mitchell et al., 1977; McCloskey & Mitchell, 1972; Strange et al., 1993). However the removal of afferent feedback by epidural anaesthesia has been shown to significantly reduce (Fernandes et al., 1989; Strange et al., 1993) or abolish (Kjær et al., 1994) this response. During dynamic exercise in

AB people the arterial baroreflex 'resets' to a higher functioning blood pressure (BP) than at rest (Papelier et al., 1994), preventing the suppression of an increase in BP (Rowell, 1974). It has been suggested that this response is mediated by central command (Rowell, 1974) or by the peripheral reflex response (Strange et al., 1993; Potts & Mitchell, 1998). BP does not increase in response to FES exercise in paraplegics (Kjær et al., 1994; Raymond et al., 2000; Dela et al., 2003). In this situation the baroreflex attempts to diminish rising BP at the onset of exercise via a reduction in HR. Consequently an initial decrease (Raymond et al., 2000) or slow rise (Dela et al., 2003) in HR at the onset of exercise has been demonstrated in paraplegics compared with AB people.

Adams et al. (1984b) reported smaller increases in $\dot{V}O_2$, $\dot{V}CO_2$, V and tidal volume (VT) in response to ES exercise in SCI people compared with AB people exercising voluntarily due to the lower work rate during ES exercise. However, greater cardiovascular, metabolic and ventilatory responses have been observed during ES than voluntary exercise at similar work rates (Strange et al., 1993). Blood pressure, leg blood flow, oxygen and glucose uptake and lactate release were greater during ES exercise, indicating a different pattern of fibre recruitment and muscular involvement. SCI people have also been reported to have a significantly greater rise in end-tidal pressure of CO_2 (Adams et al., 1984a).

6.1.2 SUBSTRATE METABOLISM

The release of hormones such as growth hormone (GH), catecholamines and adrenocorticotropin hormone (ACTH) stimulate the mobilisation and utilisation of glucose and free fatty acids (FFA) during exercise. Partial neuromuscular blockade has been shown to result in significant increases in concentrations of catecholamines, GH and ACTH during voluntary exercise in AB people (Kjær et al., 1987). Similar increases in catecholamines and GH have been noted during voluntary exercise in AB persons with sensory epidural blockade (Kjær et al., 1989). Release of ACTH was abolished in experiments with epidural anaesthesia (Kjær et al., 1989) indicating that afferent feedback is required to mediate these responses. During ES exercise in SCI people the increases in GH and catecholamines and decreases in insulin seen in AB people working voluntarily are abolished (Kjær et al., 1996b). Vissing et al. (1994) noted a significant increase in ACTH, glucose concentration and production by stimulation of group III and IV muscle afferents, which transmit information from metaboreceptors in muscle, indicating peripheral reflex control of these mechanisms (Vissing et al., 1994). During ES of paralysed skeletal muscle, glucose uptake by the muscle does not appear to be impaired (Chillibeck et al., 1999).

AB subjects exercising voluntarily and using ES at similar $\dot{V}O_2$ and HR values have shown a significantly greater whole body glucose uptake (Hamamda et al., 2004), peripheral glycogen depletion (Kim et al., 1995b; Kjær et al., 1996a) and carbohydrate (CHO) oxidation (Hamada et al., 2004) during ES induced exercise.

Similarly, a reduction in plasma [glucose], [FFA] and FFA appearance rate has been observed in SCI people during ES but not in SCI or AB people exercising voluntarily at comparable $\dot{V}O_2$ rates (Kjær et al., 1996b; Kjær et al., 2001b).

A greater build up of lactate and hydrogen ions has also been noted in AB people during ES than voluntary exercise (Kim et al., 1995; Hamada et al., 2004). These are indicative of a greater contribution of anaerobic pathways to energy production during ES exercise due to the preferential recruitment of type II fibres during ES (Sinacore et al., 1990). Since SCI is known to alter the physiological, morphological and contractile properties of type I fibres towards that of type II (Salmons & Sreter, 1976; Salmons & Henriksson, 1981; Eisenberg & Salmons, 1981; Hudlicka et al., 1982; Eerbeek et al., 1984), it is likely this affect will be more pronounced in SCI than AB people.

Overall, people with SCI show considerable inefficiency when carrying out ES exercise as indicated by very low power outputs with relatively high metabolic cost (Kjær et al., 1994). Power output has been shown to be a third lower during exercise with ES than AB people working voluntarily at the same $\dot{V}O_2$ (Kjær et al., 1994). This limiting factor is possibly due to:

i) Consequences of ES such as preferential type II fibre recruitment, and synchronous firing of motor units resulting in a greater anaerobic contribution to exercise and thus high levels of fatigability.

ii) Consequences of SCI such as reduced HR and BP responses to exercise, venous pooling, large relative contributions of fast twitch muscle fibres resulting in a greater anaerobic contribution to exercise and impairments in the mobilisation of glycogen and FFA's.

Together these factors might contribute to the very low power outputs that have been observed during ES induced exercise for SCI people. By monitoring metabolic responses during FES exercise in SCI people, this chapter aimed to identify some of the factors that cause changes in power output during FES. This information can be used to adapt and improve current FES protocols in terms of stimulation parameters.

6.2 METHODOLOGY

Two additional tests were carried out after training in order to further investigate the low power outputs despite a one year FES cycling programme that resulted in significant improvements in quadriceps muscle size, strength and fatigue resistance when tested using unilateral isometric contractions.

6.2.1 PEAK POWER TEST

Following a one year FES training programme the 5 SCI subjects described previously (Chapter 2) completed a 10 minute peak power exercise test to assess peak power output, fatigability and any subsequent recovery during FES cycling. Electrode positioning and stimulation parameters were as previously described (Chapter 2). The test commenced with 4 minutes passive cycling. Stimulation intensity was then ramped within 5 seconds to 100% and remained at this level for

10 minutes. Power was then reduced to the lowest stimulatable work rate (passive power + 1 W) for a 5-minute recovery period. Breath-by-breath $\dot{V}O_2$, $\dot{V}CO_2$, V and RER were monitored continuously at rest, during passive cycling and through out the 10 minute stimulation period using the Metamax 3B (as described previously, Chapter 4). Power output was measured by force at the crank and cadence. Cadence was maintained at 50 rpm by a motor controller (as described previously, Chapter 4).

Breath-by-breath $\dot{V}O_2$, $\dot{V}CO_2$, V, end-tidal CO_2 and RER were time-locked with power output data using custom designed software (Microsoft excel, Christopher, 2006, unpublished). Peak and lowest power output was measured for each subject. Percentage change in power output from the lowest power was also measured at 5 and 10 minutes into the test.

Analysis of variance (ANOVA) was used to assess whether power output, $\dot{V}O_2$, HR, V and RER had changed significantly during the 10 minute tests. Post hoc analysis was carried out using paired Students T-Tests to identify significant changes.

6.2.2 HOME TRAINING SIMULATION

Following the one year FES training programme, 4 out of the 5 SCI subjects (data for Subject 1 was missing due to equipment failure) completed a laboratory training session on a trike similar to their own (Fig. 7.1) in order to assess metabolic responses during training sessions. Subjects were asked to carry out a one hour

training session in exactly the same way as they did at home. Cadence and power output were recorded manually at 5 minute intervals throughout the test. Breath-by-breath $\dot{V}O_2$, $\dot{V}CO_2$, V and RER were monitored continuously at rest and through out the one hour training session using the Metamax 3B as described previously (Chapter 4). Subjects were allowed to remove the mask during the test at their request. Power output was measured by resistance on the back wheel set by the ergotrainer as described previously (Chapter 2) and monitored at 5 minute intervals.

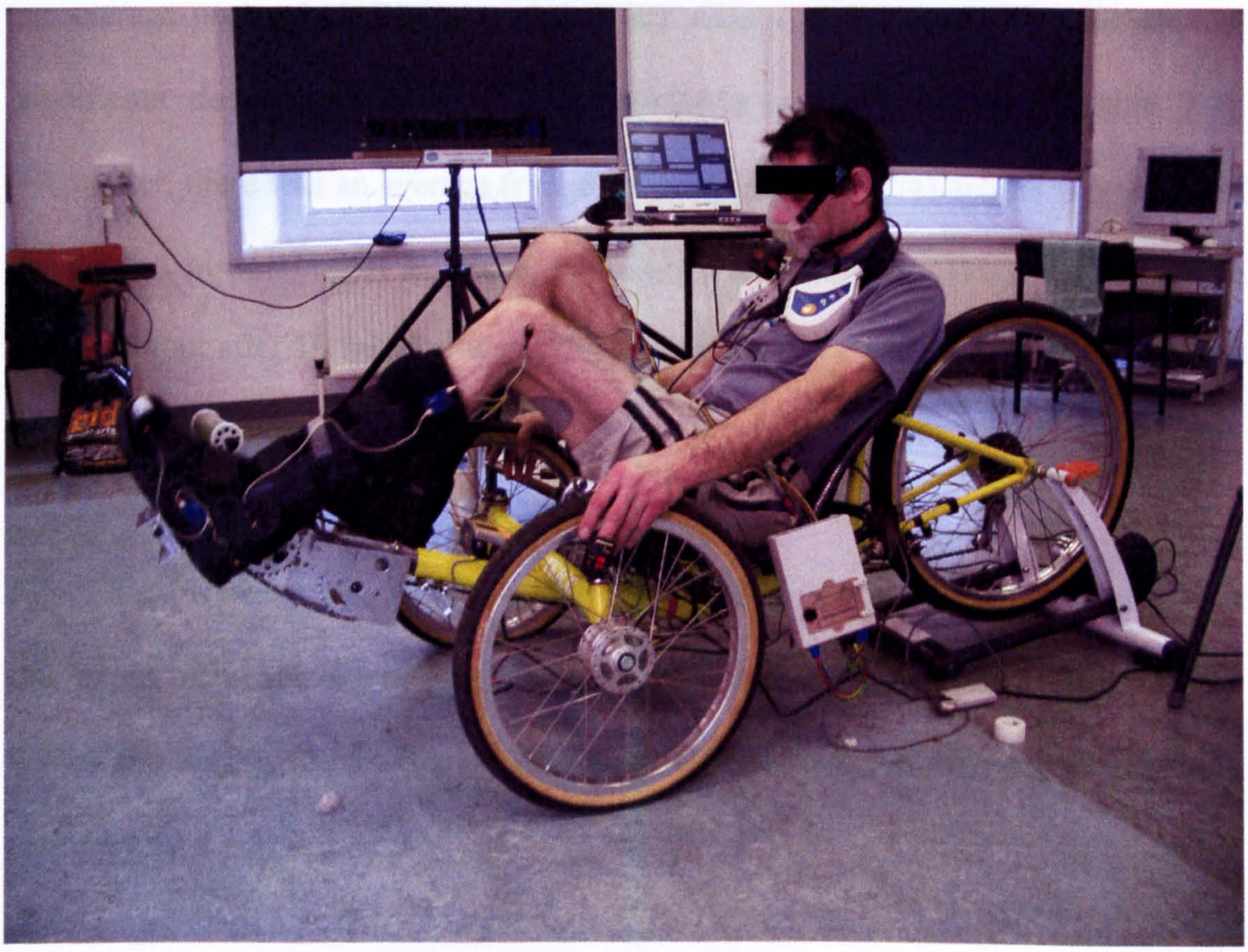

FIG 6.1: SIMULATED TRAINING SESSION WITH METABOLIC MEASUREMENTS USING THE METAMAX 3B.

Data collected when the mask was not being worn were removed from each data set. 1-3 minutes of steady state rest data were averaged and minute-by-minute data was averaged for the 1 hour training session. Data sets from the 4 subjects were then averaged and time-locked with averaged power output data.

6.3 RESULTS

6.3.1 PEAK POWER TEST

All subjects showed a initial significant increase in power output ($P < 0.01$) reaching an average peak of 49 W (range 33-85 W) within 2-3 minutes with a subsequent significant decline in power output ($P < 0.01$) to 15-30 % peak (Table 7.1). Thereafter, there was an average (range) increase of 39 (19-64) and 57 (26-99) % of the lowest power output at 5 and 10 minutes, respectively (Table 7.1). Average power output after 10 minutes was 17 W (range 9-32 W), which was similar to peak power attained during the IET (10-37 W).

Fig. 7.2 shows examples from 2 subjects of power output responses. It is clear that the extent of power output recovery varies between subjects with Subjects 2 (Fig. 7.2 a) and 5 (Fig. 7.2 b) increasing by 78 and 26 % of the lowest power output, respectively.

Subject	Peak Power (Watts)	Lowest Power (Watts)	5 min		10 min	
			Watts	% low power	Watts	% low power
1	32.6	6.6	10.9	64.1	13.1	97.8
2	40.6	9.0	13.5	50.6	16.0	78.4
3	42.8	10.2	8.6	29.3	8.8	46.7
4	42.8	6.4	13.1	33.0	14.9	36.1
5	84.5	25.2	29.9	18.5	31.7	25.8
Average	**48.7**	**11.5**	**15.2**	**39.1**	**16.9**	**57.0**
SEM	**9.1**	**3.5**	**3.8**	**8.1**	**3.9**	**13.5**

TABLE 6.1: PEAK AND LOWEST POWER OUTPUT (WATTS) AND PERCENTAGE CHANGE IN POWER FROM LOWEST POWER AT 5 AND 10 MINUTES.

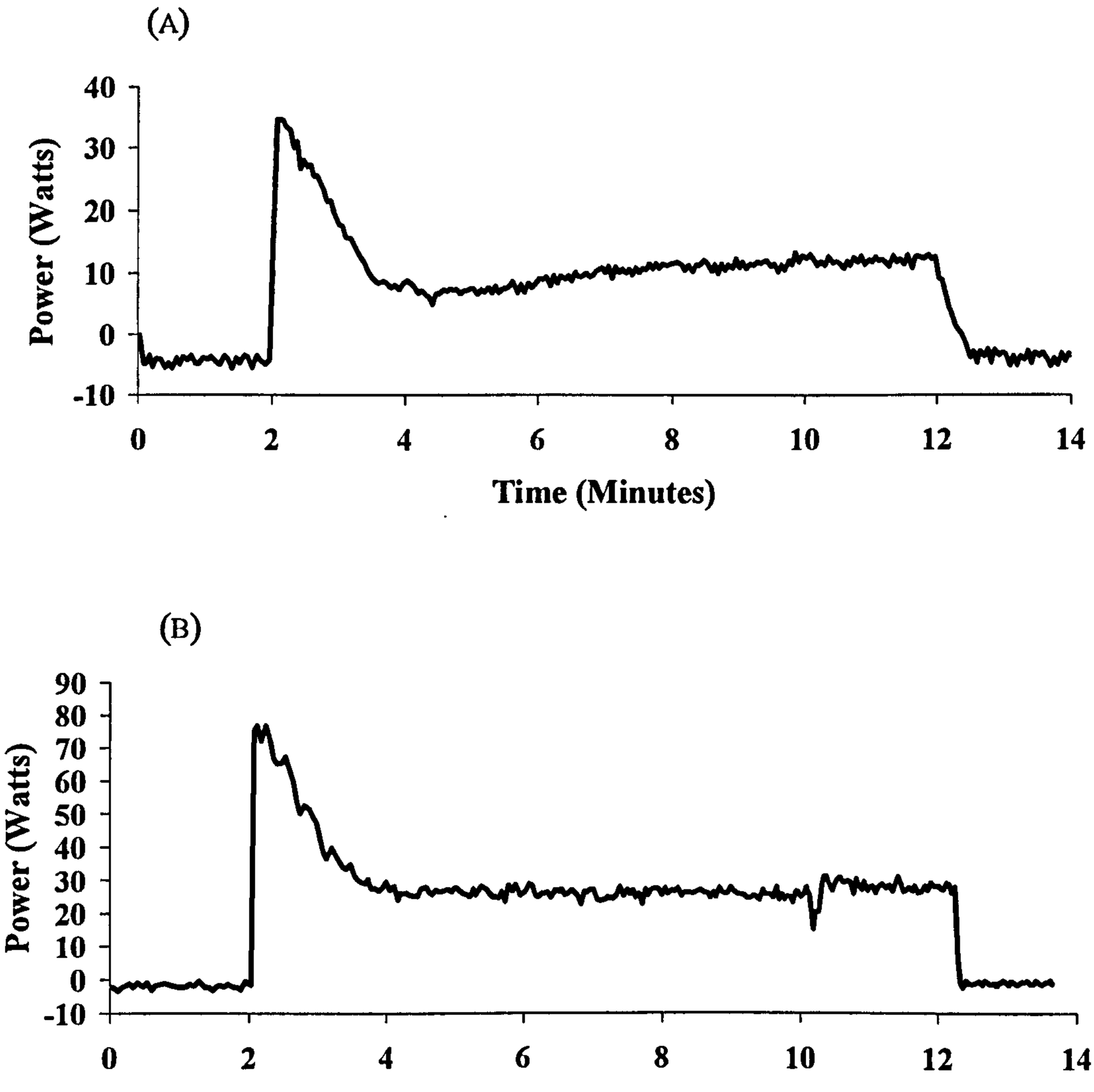

FIG. 6.2: POWER OUTPUT FOR 2 SUBJECTS (A = SUBJECT 2, B = SUBJECT 5), DURING 10 MINUTES CYCLING AT 100 % STIMULATION INTENSITY.

$\dot{V}O_2$ and HR increased to 96 and 98 % peak $\dot{V}O_2$ and HR respectively by 3 minutes and remained at this level for the rest of the test (Figs. 6.3 and 6.4). V increased to 85 % peak (assessed by IET, see Chapter 4) by 3 minutes and continued to gradually increase reaching 110 % peak at 10 minutes (Fig 7.5). RER attained a peak of 1.25 at 3 minutes and subsequently gradually declined but remained > 1.0 throughout the test (Fig 6.6).

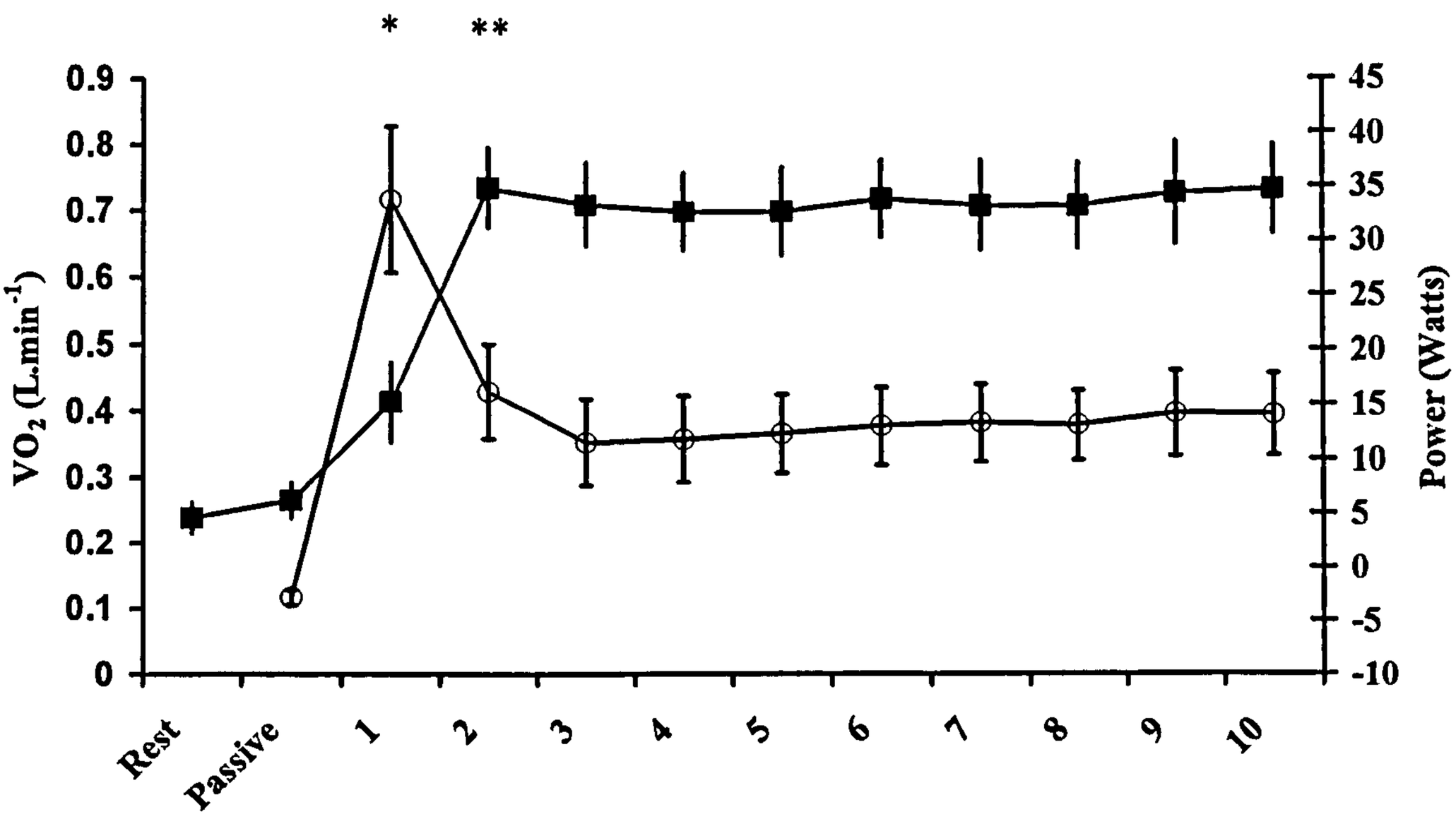

Fig 6.3: AVERAGE POWER OUTPUT (OPEN CIRCLES) AND OXYGEN UPTAKE ($\dot{V}O_2$, CLOSED SQUARES) IN RESPONSE TO 10 MINUTES FES CYCLING AT 100 % STIMULATION INTENSITY (OXYGEN UPTAKE SIGNIFICANTLY DIFFERENT THAN PRECEEDING POINT (*P<0.05, **P <0.01)).

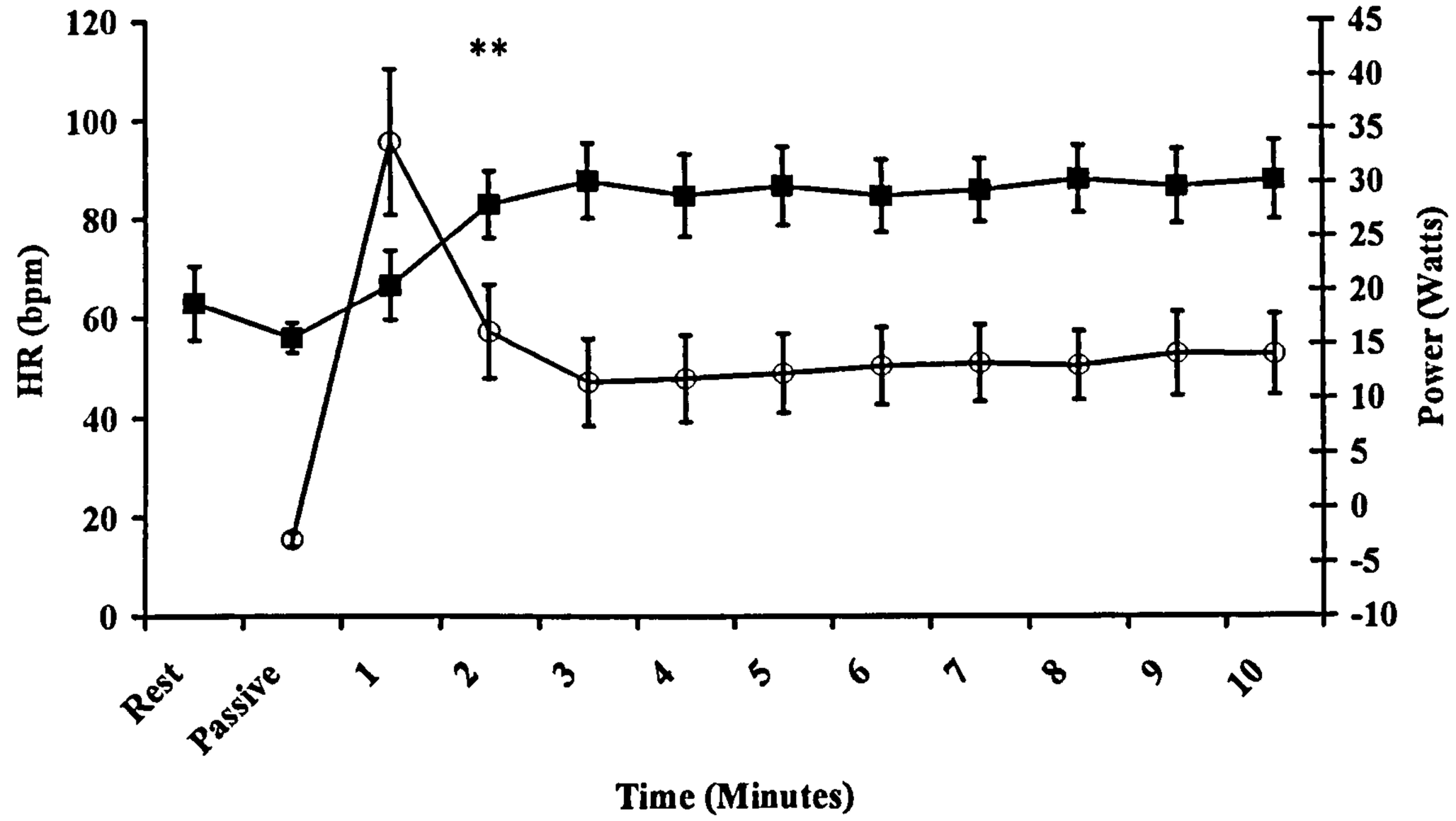

FIG 6.4: AVERAGE POWER OUTPUT (OPEN CIRCLES) AND HEART RATE (HR), CLOSED SQUARES) IN RESPONSE TO 10 MINUTES FES CYCLING AT 100 % STIMULATION INTENSITY (HR SIGNIFICANTLY DIFFERENT THAN PRECEEDING POINT (**P <0.01)).

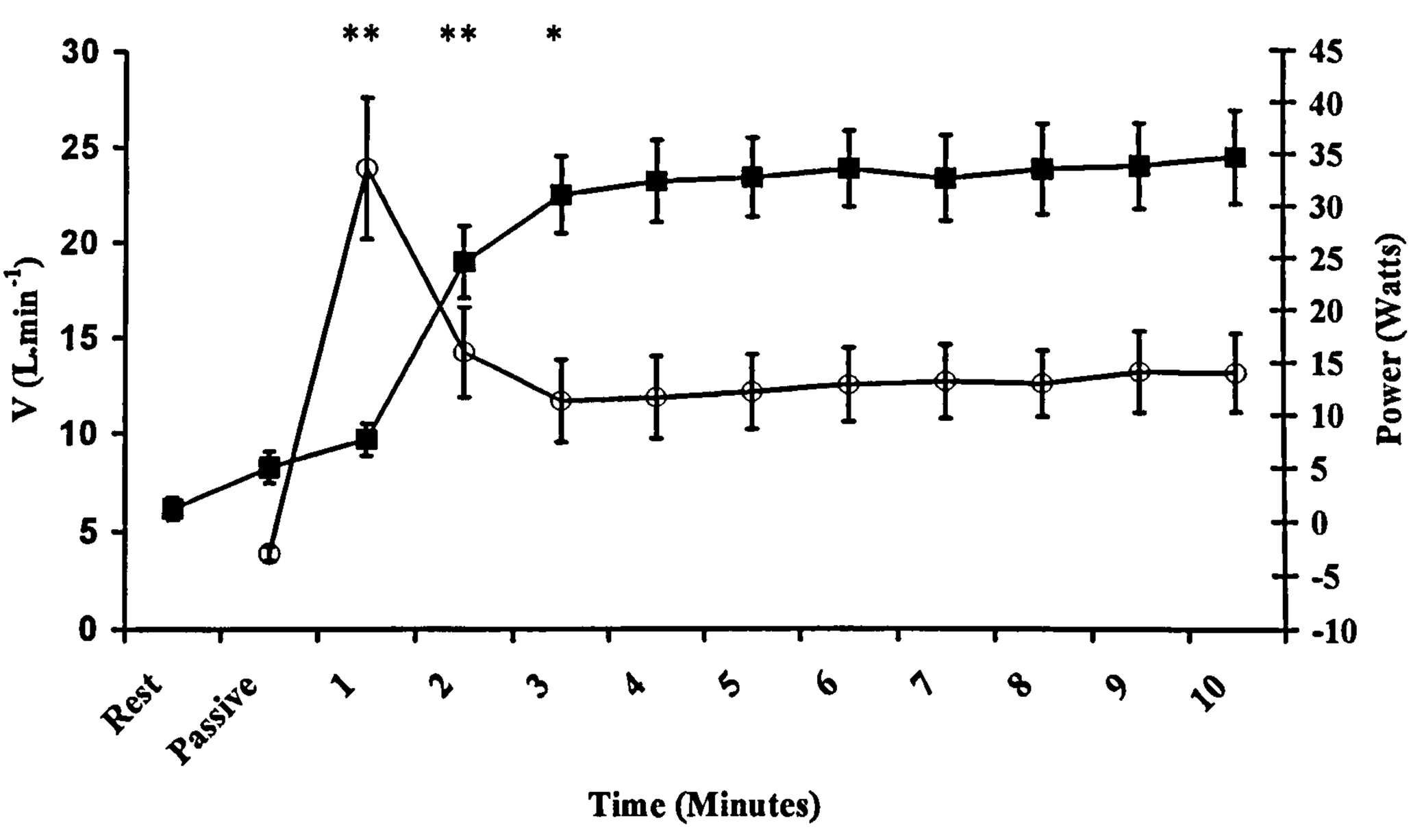

FIG 6.5: AVERAGE POWER OUTPUT (OPEN CIRCLES) AND VENTILATION (V, CLOSED SQUARES) IN RESPONSE TO 10 MINUTES FES CYCLING AT 100 % STIMULATION INTENSITY. (V SIGNIFICANTLY DIFFERENT THAN PRECEEDING POINT (*P<0.05, **P <0.01)).

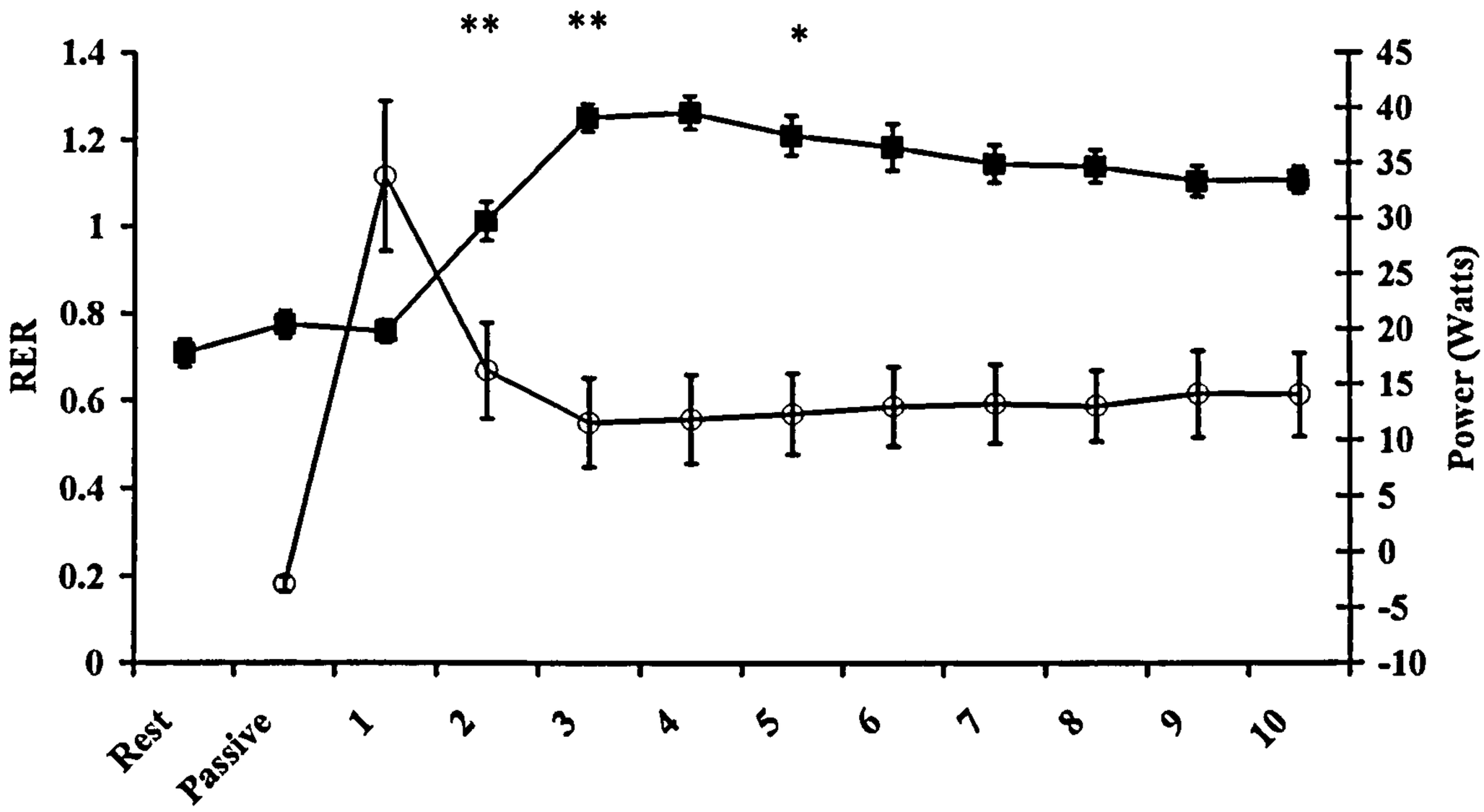

Fig 6.6: AVERAGE POWER OUTPUT (OPEN CIRCLES) AND RESPIRATORY EXCHANGE RATIO (RER, CLOSED SQUARES) IN RESPONSE TO 10 MINUTES FES CYCLING AT 100 % STIMULATION INTENSITY. (RER SIGNIFICANTLY DIFFERENT THAN PRECEEDING POINT (*P<0.05, **P <0.01)).

6.3.2 HOME TRAINING

Fig. 6.7 shows $\dot{V}O_2$, $\dot{V}CO_2$ and power output during a typical one hour training session. $\dot{V}O_2$ attained a steady state by 5 minutes at 98 % peak $\dot{V}$ O_2 (assessed by IET, see Chapter 4). Subsequently it remained at 97-103 % of peak values until 30 minutes into the training session. $\dot{V}CO_2$ attained a peak at 6 minutes into the session and subsequently declined to a minimum of 0.58L.min^{-1} at 35 minutes. The decline in $\dot{V}CO_2$ appeared to correlate with the decline in power output from 13.8 W at 1 minute to 5.5 W at 35 minutes. After 35 minutes, power output recovered to 8 W and $\dot{V}O_2$ and $\dot{V}CO_2$ also gradually increased toward the end of the session.

Between 5 and 35 minutes into the session V was on average 114 % V peak (assessed by IET, see Chapter 4). Subsequently it declined but remained 88-110 % peak V for the rest of the hour session (Fig. 6.8).

Respiratory exchange ratio (RER) reached a peak of 1.3 at 4 minutes and subsequently declined reaching a value <1.0 by 33 minutes (Fig. 6.9). The decline in power output appeared to be correlated to the decline in RER. As power output gradually improved from 35-60 minutes RER remained < 1.0 (0.88-0.99).

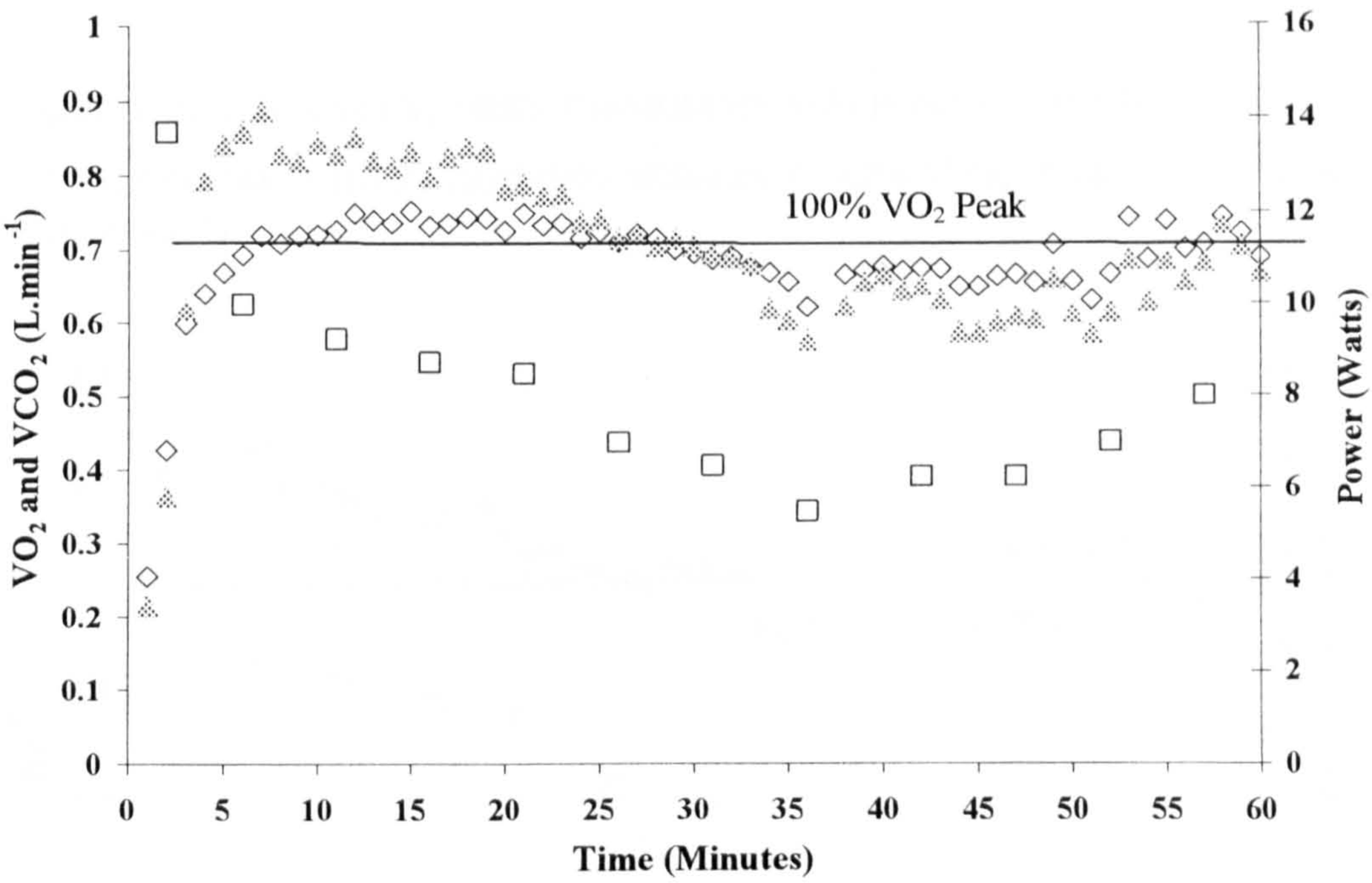

Fig. 6.7: OXYGEN UPTAKE ($\dot{V}O_2$, OPEN DIAMONDS), CARBON DIOXIDE OUTPUT ($\dot{V}CO2$, GREY TRIANGLES) AND POWER OUTPUT (OPEN SQUARES) DURING A TYPICAL ONE HOUR TRAINING SESSION. (100% $\dot{V}O_2$ PEAK ASSESSED BY IET, SEE CHAPTER 4).

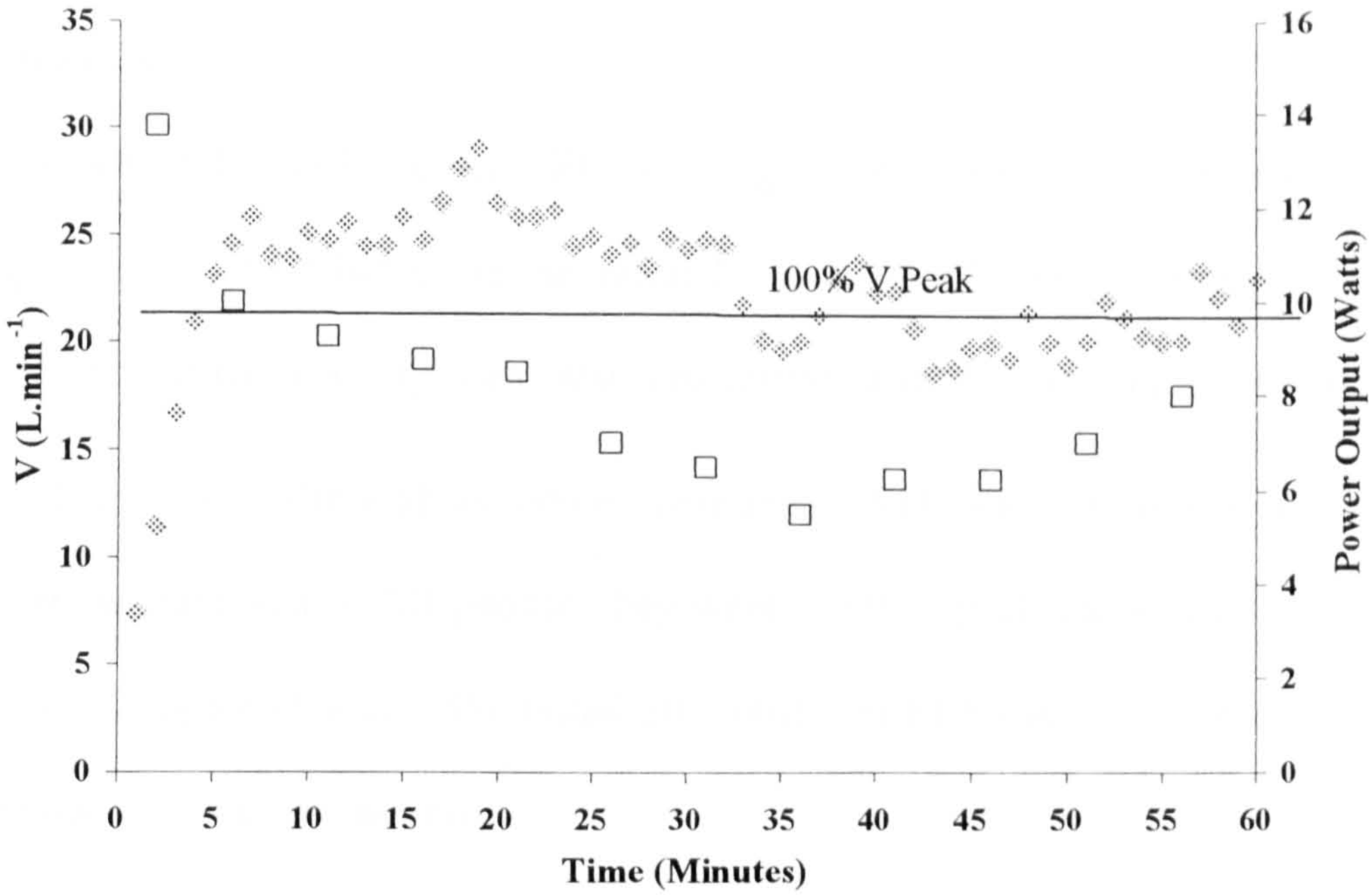

FIG. 6.8: VENTILATION (V, GREY DIAMONDS) AND POWER OUTPUT (OPEN SQUARES) DURING A TYPICAL 1 HOUR TRAINING SESSION. (100% $\dot{V}O_2$ PEAK ASSESSED BY IET, SEE CHAPTER 4).

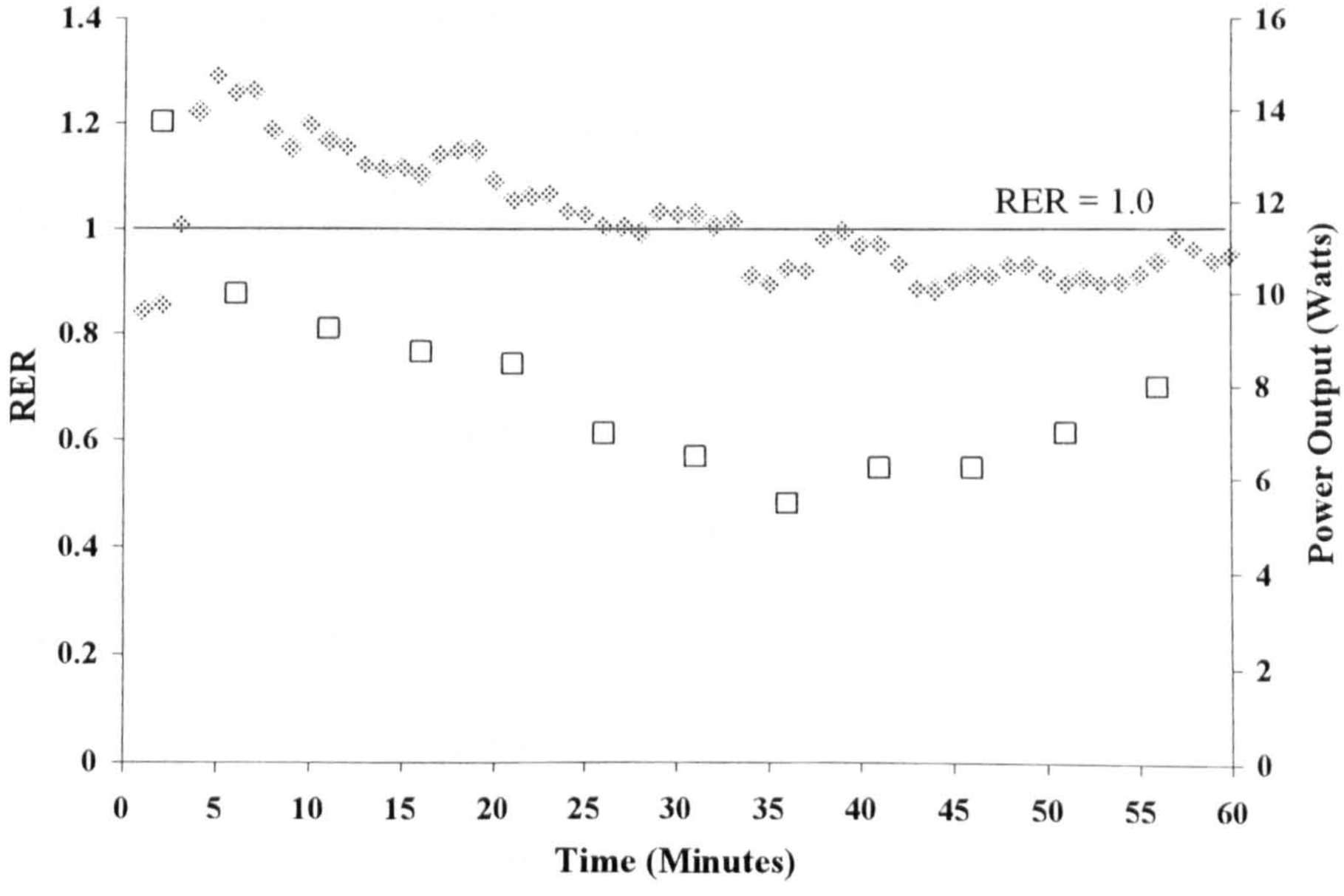

FIG. 6.9: RESPIRATORY EXCHANGE RATIO (RER, GREY DIAMONDS) AND POWER OUTPUT (OPEN SQUARES) DURING A TYPICAL 1 HOUR TRAINING SESSION.

6.4 DISCUSSION

Power output during 10 minutes FES cycling at 100 % stimulation intensity showed a rapid and severe reduction in the initial 2-3 minutes followed by a partial recovery. A second partial recovery was observed during a one hour training simulation at ~30-35 minutes. Although metabolic responses (VO_2 and V) were low compared with those observed in AB people, they were >100 % peak values (measured by an IET, see Chapter 4) during the initial 30 minutes of FES cycling. Additionally RER remained >1.0 during this time.

6.4.1 PEAK POWER TEST

After an initial mean peak of 49 W at approximately 9 s, power output declined to 11 W and subsequently recovered to 15 and 17 W at 300 and 600 s respectively. The peak power attained was significantly lower than that achieved by AB people (250-400 W). The power output at the end of the test (17 W) was also substantially lower than would be expected from AB people during voluntary steady state exercise and would create only just enough power to cycle on a flat smooth surface. It is interesting to note that SCI people appeared to reach a steady state at 10 minutes that was similar to the peak power output achieved during the IET after 12 months training (Chapter 4). AB people achieve considerably higher power outputs during a $\dot{V}O_2$ max test (250-400 W), but this can not be maintained at a steady state.

The rapid and pronounced decline in power output during the initial 30 seconds of the power test was similar to the anaerobic fatigue that occurs in AB people e.g. during the Wingate test. It is likely that the intramuscular high-energy phosphates adenosine triphosphate (ATP) and phosphocreatine (PCr) fuelled the initial few seconds of exercise and subsequently anaerobic glycolysis generated increasingly more energy for ATP resynthesis, resulting in the rapid decline in power output. A strong correlation between the reduction of power output and the profiles of PCr, Pi and intracellular pH has been reported in SCI people exercising by means of FES (Mizahri et al., 1997).

It has been widely suggested that muscular acidosis directly impairs the contractile process or the capacity of the muscle to regenerate ATP (Hermansen, 1981). In vitro studies have suggested that power output (Godt & Nosek, 1989; Cooke et al., 1988), Ca^{2+} sensitivity (Godt & Nosek, 1989), ATPase activity and maximum velocity of shortening (Cooke et al., 1988) are decreased with increased $[H^+]$ and P_i or reduced pH. More recently it has been shown that increased acidity *per se* does not cause a reduction in force generating capacity (Bangsbo et al., 1992). Pate et al. (1995) showed that, at temperatures which are comparable to those seen in mammalian skeletal muscle, muscle acidosis had minimal effects on power output and Vmax. Furthermore, following 30 seconds exhaustive exercise and a recovery period of 3 minutes, restoration of peak power output has been noted despite no recovery of the reduced muscle pH (Bogdanis et al., 1995).

Alternatively, since FES cycling was carried out at a stimulation frequency of 50 Hz, the rapid decline in power output to 22 % of the initial might have been the result of high frequency fatigue (HFF) (Jones, 1981; 1996). Low frequency stimulation immediately following high frequency stimulation has been shown to result in a rapid improvement in force generation (Jones, 1981) indicating that the cause of fatigue is post-synaptic, involving increased extracellular potassium (K^+) concentration (Bigland-Richie et al., 1979; Jones, 1981) and a slow inactivation of the sodium (Na^+) channels (Ruff et al., 1987). A rapid recovery in power output during FES cycling was observed following a short pause (10 seconds) in stimulation (data not presented) suggesting that HFF is at least part of the cause of the initial rapid decline in power output. It is therefore possible that reducing the firing frequency after the initial few impulses would prevent such a severe decline in power output at the start of exercise. Alternatively, recruitment of additional or alternative motor units might reduce this severe fatigue.

Following an initial 2 minutes of all-out exercise in AB people, the relative contribution of anaerobic glycolysis to energy supply reduces and aerobic energy system provides the largest contribution. The rapid rise in V and $\dot{V}O_2$ in SCI people at the start of exercise (Figs. 6.5 and 6.3) indicates that there is an increasing aerobic contribution to exercise and that part of the increased drive to breathe comes from an increase in arterial CO_2 pressure and $[H^+]$ and possibly from a build up of K^+ ions (Patterson et al., 1992). From 4 minutes onwards, V remained >100% peak (assessed by an IET, see Chapter 4), suggesting that in continuing conditions of high $[K^+]$

there is a further stimulus to breathe. This is possibly hyperkalaemia combined with hypoxia (Paterson et al., 1992) considering that exercise was anaerobic (RER >1.0).

Exercise remained highly anaerobic throughout the test and exercise was fuelled by 100 % CHO metabolism as indicated by an RER >1.0 (Fig. 6.6). It has been shown that BP does not increase in response to FES exercise in SCI people (Kjær et al., 1994; Raymond et al., 2000; Dela et al., 2003), which might limit blood flow and therefore oxygen supply to muscle. Greater whole body glucose uptake (Hamamda et al., 2004), peripheral glycogen depletion (Kim et al., 1995b; Kjær et al., 1996a) and CHO oxidation (Hamada et al., 2004) has previously been reported to occur during ES exercise. This might be due to the high proportion of fast twitch fibres in SCI people. Additionally, synchronous stimulation at a high frequency would further promote anaerobic exercise resulting in muscle acidosis.

The partial recovery in power output which occurred between 2 and 10 minutes (Table 6.1) has not been reported in AB people exercising voluntarily. The reason for this recovery is unclear. It is possible that the large anaerobic contribution to exercise resulted in an increase in lactate and hydrogen ions in the muscle and muscle acidosis (Hermansen, 1981). Consequently, the reduction in the activity of glycolytic enzymes (phosphorylase and phosphofructkinase) would lead to a reduced rate of glycolysis and reduced ATP resynthesis (Hermansen, 1981). The observed fatigue might have allowed removal of lactate and H^+ and therefore the partial recovery in power output. However, it has been shown that a recovery of power

output occurs in healthy individuals despite raised levels of blood lactate (Bogdanis et al., 1998). As this type of recovery in power output has not been observed in AB people exercising voluntarily it is unlikely that the removal of lactate exclusively induced the observed recovery.

When AB people exercise voluntarily, fatigue is associated with a slowing of motor neurone firing and cycling of active motor units occurs (Carpentier et al., 2001). These mechanisms counteract the intramuscular accumulation of K^+ ions. FES cycling involves repeated activation of the same motor units at a high firing frequency (50 Hz) and thus it is possible that the continuation of exercise, despite a very high extracellular K^+ concentration, causes power output to become very low. This severe fatigue might have resulted in some muscle fibres becoming inactive, allowing potassium ions to be taken up by these inactive fibres resulting in the observed recovery in power output. This mechanism might occur in SCI people only because the movement of potassium ions has also been associated with pain in AB people (Patterson et al., 1990) resulting in central fatigue whereas SCI people have a complete lack of sensory feedback.

The recovery in power output occurred to a greater extent in some SCI subjects than others (Fig. 6.2). It is interesting to note that those who experienced the greatest recovery (Subjects 1 and 2) were the most fatigue resistant during repeated contractions of the quadriceps muscle (see Chapter 3). The least fatigue resistant subjects experienced the least recovery (26-47 %) and produced the highest instant

peak power (43-85 W compared with 33-41 W). This suggests that the subjects with the most recovery had a greater proportion of slower muscle fibres, increased capillary density or improved mitochondrial function.

It has been shown that fast fibres are more susceptible to the inactivation of the inward Na^+ current, which occurs when the action potential membrane is partly depolarised (Ruff & Whittlesey, 1992). Therefore, the recovery of the Na^+ inward current may have occurred to a lesser extent in individuals with faster muscle types resulting in a smaller recovery in power output. Muscle biopsy samples would be necessary to accurately determine muscle fibre types.

An alternative possibility is that habituation of spinal reflexes (Dimitrijević & Nathan, 1970) occurred during ES, resulting in the partial recovery in power output. FES elicits both cutaneous and muscular afferent feedback resulting in spinal reflex activity. It has been reported that spinal reflexes in SCI people are reduced by ES although responses have been reported to be variable (Andrews et al., 1989; Gregorič, 1998; Knikou & Conway, 2002; 2005). During FES cycling, it is possible that flexion reflexes occurred and contributed to the initial rapid fatigue by counteracting the production of power. Indeed, electrical stimulation induced spasms in SCI people, as observed during tests of muscle force and during FES cycling exercise tests (unpublished observations). Had these reflexes subsequently reduced or habituated this might have contributed to the observed partial recovery.

6.4.2 HOME TRAINING

Power output reached a peak at one minute into the hour training session and gradually declined to 40 % of peak by 35 minutes and subsequently recovered to 56 % after one hour. $\dot{V}O_2$, $\dot{V}$ CO_2 and V and increased rapidly at the start of exercise (Fig. 6.7 and 6.8) and subsequently declined and increased along with power output. RER increased rapidly at the start of exercise and subsequently declined to <1.0 at 35 minutes and remained <1.0 for the rest of the session (Fig. 6.9).

$\dot{V}$ O_2 increased to 0.71 $l.min^{-1}$ at 5 minutes (97% peak $\dot{V}$ O_2). Concurrently $\dot{V}$ CO_2 and RER increased, reaching peaks of 0.89 $L.min^{-1}$ and 1.29, respectively, indicating a high anaerobic contribution to the initial 5 minutes of exercise, as would be expected from AB people exercising at 100 % physical capacity. During the initial 35 minutes of training, $\dot{V}O_2$ remained at approximately 98-101% of the peak $\dot{V}O_2$ and concurrently RER remained >1.0. This is surprising considering that in AB people after the initial 5 minutes of exercise oxidation of muscle glycogen stores can normally sustain exercise at a power output equivalent to approximately 70 % $\dot{V}O_2$ max depending on training status. A combination of limited central and/or peripheral $\dot{V}O_2$, central and/or peripheral fatigue and pain would probably prevent an AB individual from exercising at maximum capacity (98-100 % peak $\dot{V}O_2$) for such a long duration. SCI people using FES exercise might be able to perform work at peak capacity for longer duration because i) absolute peak $\dot{V}O_2$ values are very low compared with AB people, thus presumably central $\dot{V}O_2$ and fatigue does not limit

performance and ii) complete sensory loss in the exercising muscles means that the build up of metabolic by-products does not induce painful sensations, thus attenuating the affects of central fatigue. Additionally, high proportions of fast twitch fibre types, synchronous recruitment of motor units and high frequency stimulation would further promote anaerobic activity. The concurrent gradual decline in PO (Figs. 6.7-6.9) suggests that the build-up of waste products such as lactate limited cross bridge formation and contractile force.

After 35 minutes exercise there appeared to be a paradoxical recovery in PO. The recovery time appeared to correlate with RER reaching a value <1.0, thus there is a greater aerobic contribution to energy supply. The greater aerobic contribution might allow additional oxygen to become available for the removal of waste products such as lactate, resulting in improved cross bridge formation and possibly the observed recovery of PO.

It is recommended that measurements of PCr, Pi and intracellular pH as well as concentrations of K^+ and lactate in the blood be taken in future tests of fatigue during FES cycling in order to gain a more complete understanding of the mechanisms resulting in fatigue, as observed in the present chapter.

6.5 Conclusions

Power output during FES cycling remained low compared with AB people, even after an intense one year training programme. There appears to be a rapid loss of

power during the initial two minutes of exercise, which might be due to high frequency fatigue, particularly given the high level of fatigue resistance found during repeated contractions of the quadriceps muscle after training (see Chapter 3). The subsequent partial recovery in power output might be due to fatigue allowing removal of potassium ions or to the habituation of flexion reflexes. In AB people, reductions in firing frequency and cycling of active motor units counteract this fatigue to some extent. Therefore, altering firing frequency and also allowing some asynchronous firing might reduce this effect.

SCI people appeared to work anaerobically for 35 minutes during a one hour training session. The reduction in RER <1.0 correlated with a second recovery in power output. Alterations in firing frequency and the use of variable frequency trains might allow a greater aerobic contribution during FES exercise. Additionally, asynchronous firing of motor units might allow improvements in lactate removal. In combination, these variables might allow improved power outputs during FES cycling. Future studies should measure the concentration of metabolites in order to elucidate the underlying mechanisms.

The following chapter compares muscle recruitment patterns during upright and recumbent cycling at varying intensities with the muscle activation pattern used for FES cycling in the present study. This aimed to investigate a possible reason for low power outputs during FES cycling in SCI people.

Chapter 7 Muscle activation patterns during voluntary upright and recumbent cycling.

The work described in this chapter aimed to determine any differences in muscle activation patterns between upright and recumbent cycling at varying cycling intensities in AB people and to compare these with currently used stimulation timings during FES cycling for SCI people. Activation timings used for current FES cycling systems are based on upright cycling, which is possibly different to activation timings during recumbent cycling. Furthermore these patterns might change with increasing cycling intensity. AB people presumably activate muscle groups in the most effective pattern, therefore it is important to understand voluntary activation patterns during both upright and recumbent cycling at varying intensities.

7.1 Literature Review

During voluntarily activated cycling, muscle activation patterns are affected by body configuration (upright or recumbent), cycling cadence and resistance.

7.1.1 Upright Cycling

A great deal of literature exists concerning the muscle activation patterns during upright cycling with general agreement about the timing and intensity of each muscle group. This review describes positions in the crank cycle based on top dead centre (TDC) representing 0°. TDC is the position at which the pedal is at

the start of the cycle where the cyclist has one knee most flexed and the contralateral knee most extended. The power phase (0-180°) is the downward driving phase and the recovery phase (180-360°) is the upward phase returning to TDC. 180° is referred to as bottom dead centre (BDC).

7.1.1.1 Muscle activation

The gluteus maximus (GMax) has been identified as the muscle group that shows the lowest peak activity during the pedal cycle (Ericson et al., 1985) and is active during the early power phase (0-90° after TDC) acting as a hip extensor (Jorge & Hull, 1986; Ericson et al., 1985; Ericson 1988; Ryan & Gregor, 1992). It has been shown to have little variability throughout the cycle, with approximate coefficient of variation (CV) values between 20 and 30 % (Ryan & Gregor, 1992). The function of the gluteus medius (GMed) muscle is however less clear (Ericson et al., 1985; Ericson, 1988). It is active during hip and knee extension (Clarys et al., 1988) but might act to prevent knee adduction or external rotation of the femur (Ericson et al., 1985; Ericson, 1988).

Rectus femoris (RF) shows lower overall activity than single joint quadriceps muscles such as vastus lateralis (VL) and vastus medialis (VM) (Ericson et al., 1985). It is active for a large proportion of the crank cycle beginning at the middle-end of the recovery phase and continuing until the middle-end of the power phase (Carlsöö & Molbech, 1966; Jorge & Hull, 1986) indicating its role in hip flexion followed by knee extension. Peak activity of RF has been shown to occur later than VL and VM during the power phase, suggesting it is most important as a knee extensor (Carlsöö & Molbech, 1966; Ericson et al., 1985).

Ryan & Gregor (1992) however showed peak RF activity to occur at the end of the recovery phase with less activity during the power phase, suggesting a more important role as a hip flexor. Its lower activity during the power phase is understandable because this would counteract simultaneous hip extension (Ericson, 1988). The variation between these studies might be due to different loads and seat heights or different techniques used by the participants, based on their cycling experience. RF has been shown to have a larger CV (28-58 %) during the entire cycle compared to the VL and VM, probably because it is a two-joint muscle (Ryan & Gregor, 1992).

VM and VL have been reported to be the most highly activated muscles during cycling and to produce the highest power (Ericson et al., 1985). Thus they can be considered as the most important of the muscle groups that contribute to the cycling motion. Approximately synchronous firing has been noted between the two muscle groups (Ryan & Gregor, 1992). The onset of their activity generally occurs later than that of RF during the knee extension phase (Jorge & Hull, 1986), continuing until approximately 130° after TDC. Their peak activity occurs approximately 30° after TDC (Ericson et al., 1985; Clarys et al., 1988; Ryan and Gregor, 1992).

The biceps femoris (BFem) appears to be active at a low level throughout the cycle (Ericson et al., 1985; Ryan & Gregor, 1992) and two distinct patterns of low-level peak activity have been identified. The first pattern shows peak activity at TDC and early into the power phase (Ericson et al., 1985; Ryan & Gregor, 1992). In the second pattern this occurs late in the power phase (approximately

70° after TDC until BDC) with decreased activity during knee flexion (Jorge & Hull, 1986; Clarys et al., 1988; Ericson, 1988; Ryan & Gregor, 1992). The second pattern indicates that BFem works as a hip extensor (Ericson, 1988) and towards the latter stages of the power phase becomes the dominant muscle at the knee joint initiating flexion after knee extensor activity decreases at approximately 130° after TDC (Clarys et al., 1988). The reason for the existence of two distinct patterns might be because of different load distribution among the hamstring muscles occurring at higher workloads (Ryan & Gregor, 1992).

The semitendinosus (ST) and semimembranosus (SM) follow similar patterns throughout the cycle with a short delay in ST activity (Ryan & Gregor, 1992). They have been reported to be active from either the early (~60° after TDC) (Jorge & Hull, 1986; Ryan & Gregor, 1992) or late (~150° after TDC) (Ericson et al., 1985) power phase until approximately 270° after TDC (Ericson et al., 1985; Jorge & Hull, 1986; Ryan & Gregor, 1992), with peak activity occurring at approximately 94 and 122° for SM and ST, respectively (Ryan & Gregor, 1992). In contrast, Ericson (1988) noted that peak medial hamstring activity (ST and SM) occurred during knee flexion. The discrepancies might be due to different recording electrode placement in these studies.

The gastrocnemius muscle has been noted to be active from the late power phase and continuing into the early recovery phase (Carlsöö and Mobech, 1966), functioning as a plantarflexor to push the pedal through the BDC position (Ericson et al., 1985) with peak activity occurring at 107° (Ryan & Gregor, 1992). In contrast Jorge & Hull (1986) noted gastrocnemius muscles to be active

earlier (45-110° after TDC), during knee extension, despite their function as knee flexors. The discrepancy might be due to different functioning of the two heads of gastrocnemius, medialis (GastM) and lateralis (GastL). Ericson (1988) noted that GastM activity increased during knee extension and decreased during knee flexion, indicating that during cycling it functions more as an ankle plantarflexor than a knee flexor. GastL however showed maximum activity during knee flexion indicating its dominant role as a knee flexor (Ericson, 1988).

The activity of the gastrocnemius muscle has been shown to be variable with CV values ranging from 30 % (during recovery phase) to 99.7 % (near TDC) (Ryan & Gregor, 1992) which might further explain the discrepancies noted above. In addition, GastM has been shown to be active earlier in sprint compared to submaximal cycling (Clarys et al., 1988).

7.1.1.2 Effect of changing cycling parameters

Houtz & Fischer (1959) noted that when no load was applied during cycling, action potentials occurred primarily in the muscles of the foot and ankle (gastrocnemius and tibialis anterior). The duration and intensity of muscular activity has been shown to increase with increasing load (Houtz & Fischer, 1959; Ericson et al., 1985; Jorge & Hull, 1986; Clarys et al., 1988). In particular, the single-joint muscles (GMax, GMed, VM and VL) show an almost linear increase in activity with power output whereas two-joint muscles (RF, BFem, ST, SM and gastrocnemius) have shown no consistent relationship with power output (Ericson, 1988). Increasing cycling cadence has been shown to significantly increase activity levels of the GMax, GMed, VM, ST, SM and GastM (Ericson et

al., 1985), although not to the same extent as with increasing load (Houtz & Fischer, 1959). The timing of muscular activity during cycling has been reported to be unaffected by the load applied (Jorge & Hull, 1986). However, Clarys et al. (1988) demonstrated increased and decreased duration of activity in BFem and gastrocnemius respectively during sprint compared to submaximal road cycling.

Varying seat height position has also been found to affect muscle activation intensity. Reducing the seat height from 100-95 % of the trochanter increased muscular activity (Jorge & Hull, 1986). Increasing the seat height from 102-120 % of the distance between the ischial tuberosity and the medial malleolus has also increased muscular activity (Ericson et al., 1985). This suggests that an optimal seat height exists, where activity levels are lowest and thus cycling is more efficient.

7.1.2 RECUMBENT CYCLING

Compared with upright cycling, recumbent cycling results in changes in leg orientation and trunk angle, which affect the contribution of gravity to the motion and the muscles that span the hip joint, respectively. Recumbent cycling also alters the extent to which body mass can be used. Very little data has been collected regarding the muscle activation patterns during recumbent cycling, however the literature that does exist suggests differences in the recruitment patterns of some muscles during recumbent and upright cycling (Trumbower & Faghri, 2004).

7.1.2.1 Muscle activation

The more horizontal trunk position has been reported to result in increased mean muscle length (over one crank cycle) of the BFem, ST, RF, tibialis anterior (TA), VM, soleus and GastL, whilst decreasing that of Gmax (Savelberg & Meijer, 2003). Alterations in muscle length would affect the muscle's ability to generate force. Indeed, this trunk position has been reported to significantly increase the average muscle activity of the BFem, ST, RF, TA, soleus and GastL, but not alter Gmax activity at a given power output (Savelberg & Meijer, 2003). Thus although alteration in trunk angle directly affects the muscles that span the hip, recruitment of all muscles in the lower limb are affected (Savelberg & Meijer, 2003).

During recumbent cycling, lowest EMG values have been noted in the gluteal muscles with peak activity occurring during the early power phase (Trumbower & Faghri, 2004), similar to upright cycling. However, the onset of activity during recumbent cycling has been shown to be just before TDC, slightly earlier than that seen in upright cycling (Trumbower & Faghri, 2004).

Activity in the quadriceps appears to be similar to upright cycling, starting at the middle-end of the recovery phase, but terminating earlier in the power phase (~90°) (Trumbower & Faghri, 2004). However data regarding the separate activity of RF, VL and VM during recumbent cycling are not presented in that study. Data during upright cycling suggests that VL and VM continue their activity further into the power phase compared to RF (Jorge & Hull, 1986)

however little data exists regarding activity of separate quadricep muscles during recumbent cycling.

The onset of hamstrings activity has been found to be during the middle part of the power phase continuing until approximately 260° after TDC. This activation pattern is later than suggested by some studies for BFem during upright cycling (Ericson et al., 1985; Ryan & Gregor, 1992). However, these timings are similar to those suggested by Jorge & Hull (1986) for ST and SM during upright cycling.

The onset of gastrocnemius activity has been reported to occur approximately 130° after TDC during recumbent cycling (Trumbower & Faghri, 2004), later than suggested during upright cycling (Jorge & Hull, 1986). However, Trumbower & Faghri (2004) measured GastL activity only, which has been shown to act primarily as a knee flexor during upright cycling (Ericson, 1988). Thus the activity of GastL during recumbent cycling is in agreement with the same muscle group during upright cycling.

7.1.2.2 Effect of changing cycling parameters

Similar to upright cycling, increased muscle activation has been noted with increased resistance during recumbent cycling (Trumbower & Faghri, 2004) and the timing of EMG activity has been reported to be unaffected by altered resistance (Savelberg & Meijer, 2003; Trumbower & Faghri, 2004).

7.2 METHODOLOGY

Eight AB people (3 female) were tested on 2 occasions at least 24 hours apart, cycling on an upright ergometer and a recumbent tricycle. All subjects were not competitive cyclists and average age, height and body mass were 40.2 ± 3.8 years, 173.6 ± 2.3 cm and 77.1 ± 6.0 kg, respectively. On each occasion subjects carried out 5 three minute periods of cycling at 12, 25, 50, 75 and 100 W, each separated by one minute rest and a one minute period of cycling at 250 W or their maximum ability (if lower). Subjects were asked to cycle continuously at 50 rpm and to maintain a consistent upper body posture and remain seated throughout each test. Crank distance from the seat was set for both upright and recumbent cycling as where knee joint angle of the extended limb was approximately 10° (protocol used for FES cycling set-up). Foot straps were used during upright cycling and during recumbent cycling subjects' feet were held in foot and ankle orthoses attached to the pedals (used during FES cycling for SCI people) which allowed no movement at the ankle joint.

7.2.1 DATA COLLECTION

The CODA motion analysis system (MPX, Charnwood Dynamics, UK) was used to track motion of body segments during cycling. Infra red emitting markers attached to the subjects' skin were tracked by the motion analysis system. EMG electrodes were also attached to the subjects' skin and EMG data was time-locked with associated motion data.

Motion Analysis: Six markers were positioned on one side of the test subject as follows: (1) fifth metatarsal, (2) heel, (3) lateral malleolus, (4) lateral side of head

of tibia, (5) greater trochanter and (6) anterior superior iliac crest. An infra red camera placed to the side of the cycle received infra red signals from each marker. The heel marker at its lowest position was set as the origin and all measures were related to that marker, therefore all values were positive. A 3D position co-ordinate was assigned to each marker at a frequency of 800 Hz. This frequency is lower than recommended, however we were limited by the equipment available. Since the data in the present study considers muscle activation patterns over a 1-2 second period (i.e. one cycle) it was decided that this sampling frequency was sufficient. A personal computer received and displayed location signals for all markers. The system was factory calibrated in the X, Y and Z planes.

Electromyography (EMG): Electrodes were positioned on 4 muscle groups (those stimulated during FES cycling) as follows:

1) Rectus femoris: 50% of the line between the pelvic crest and the superior part of the patella.
2) Biceps femoris: 50% of the line between the ischeal tuberosity and the lateral head of tibia.
3) Medial gluteus: 50% of the line between the sacrum and the greater trochanter.
4) Lateral Gastrocnemius: One-third (proximal) of the line between the head of fibula and the heel.

Data was collected for three 5 second periods at the end of each stage resulting in approximately 12 cycles of data per subject for each intensity.

7.2.2 Data Analysis

The trajectory of each marker position was plotted as shown in Fig. 7.1. One cycle was defined from one point in the signal to the next in the same position and moving in the same direction. Interpolation was then used to join data points and 100 samples were taken within each cycle, the first being the point at the start of the cycle. The EMG signal was recorded at these 100 time points for each cycle. This created a number of data sets (according to the number of cycles recorded at that cycling intensity) that contained EMG data from 4 channels and data from each marker for each cycle. EMG data were averaged and this value was then subtracted from the data to find the true zero and the data were then rectified. EMG and marker data for each of the 100 samples recorded per cycle were then averaged. Data from each marker was used to create three joint angles (hip, knee and ankle). Finally the data was organised so that the first point was the position where the knee was most flexed (TDC) i.e. at the smallest joint angle.

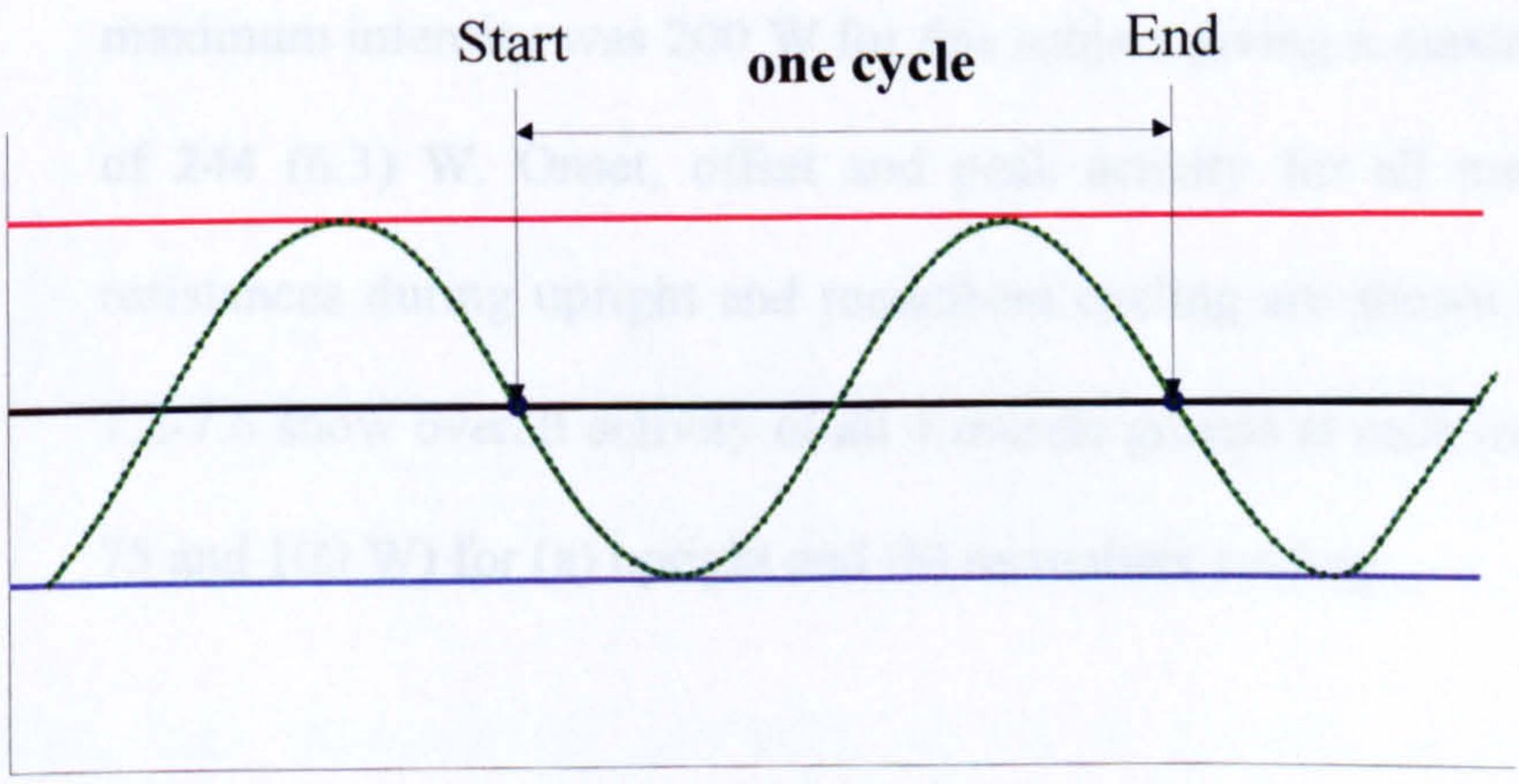

Fig 7.1: Interpolated trajectory of one marker postion (green line). Samples were taken at 100 points within one cycle. (Red, blue and black lines show minimum, maximum and mean marker position).

EMG data were expressed as a percentage of the maximum EMG signal recorded during cycling at 250 W (or maximal intensity if lower) for each muscle group during upright or recumbent cycling. Data for each muscle group at each intensity were then averaged and plotted against crank angle (where TDC = 0°). Onset and offset of muscle activation was determined for each muscle group. Since absolute resting data was not obtained in this study, muscles were considered active when activity was >10 % peak values during cycling at maximum, except for GMed during recumbent cycling, which was considered active at >20 % due to higher baseline activity (Figs. 7.2-7.6 (b)).

It was not possible to carry out statistical analysis on the data collected because averaging was carried out automatically using custom designed software (Matlab).

7.3 RESULTS

One subject was not able to attain 250 W during recumbent cycling. Therefore maximum intensity was 200 W for this subject giving a maximum (SEM) power of 244 (6.3) W. Onset, offset and peak activity for all muscle groups at all resistances during upright and recumbent cycling are shown in Table 7.1. Figs. 7.2-7.6 show overall activity of all 4 muscle groups at each intensity (12, 25, 50, 75 and 100 W) for (a) upright and (b) recumbent cycling.

	Power (W)	Upright					Recumbent				
		12	25	50	75	100	12	25	50	75	100
RF	On (°)	302	316	302	281	306	--	--	--	335	335
	Off (°)	4	356	36	40	68	--	--	--	68	65
	Amp (%)	14.4	14.3	16.6	20.8	26.7	--	--	--	13.9	16.4
	Angle (°)	338	349	356	353	11	--	--	--	4	43
B Fem	On (°)	122	97	94	90	76	158	119	94	50	36
	Off (°)	187	212	238	256	270	212	230	252	259	270
	Amp (%)	19.6	20.9	27.0	29.2	27.3	13.3	15.9	24.4	21.2	22.1
	Angle (°)	140	140	140	144	166	184	176	176	158	166
G Med	On (°)	--	--	108	32	32	--	--	--	43	40
	Off (°)	--	--	148	140	140	--	--	--	144	155
	Amp (%)	--	--	12.4	12.5	16.7	--	--	--	25.6	28.7
	Angle (°)	--	--	130	115	101	--	--	--	65	90
Gast L	On (°)	36	43	47	58	43	155	133	115	112	112
	Off (°)	292	292	295	292	292	227	256	259	284	313
	Amp (%)	26.5	32.3	26.4	29.5	29.1	14.2	14.1	17.2	21.0	21.1
	Angle (°)	205	223	187	191	202	191	191	187	191	205

TABLE 7.1: CRANK ANGLES AT THE ONSET AND OFFSET OF MUSCLE ACTIVITY, PEAK AMPLITUDE (% MAXIMUM, AMP) AND CRANK ANGLE WHERE PEAK ACTIVITY OCCURRED FOR RECTUS FEMORIS (RF), BICEPS FEMORIS (BFEM), GLUTEUS MEDIUS (GMED) AND LATERAL GASTROCNEMIUS (GASTL) DURING UPRIGHT AND RECUMBENT CYCLING AT EACH RESISTANCE (0° = TOP DEAD CENTRE, -- = NO MEASURABLE ACTIVITY).

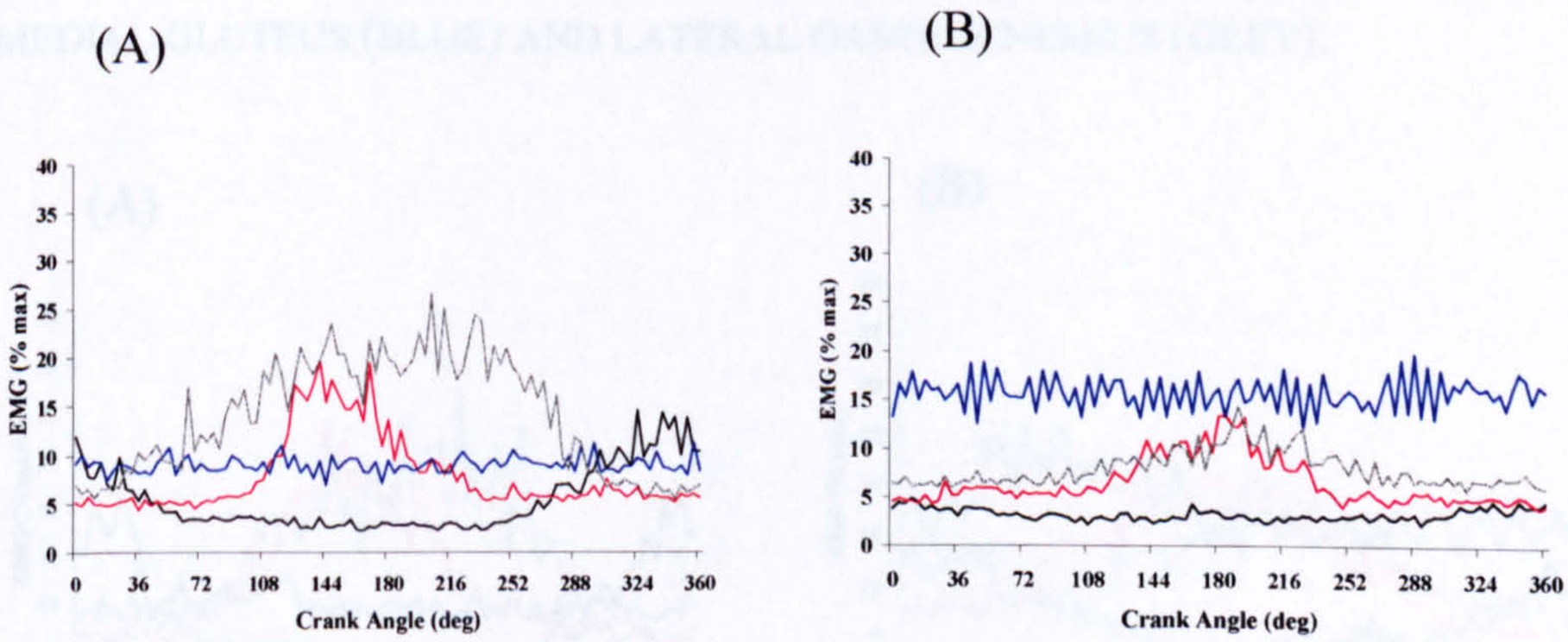

FIG. 7.2: MEAN EMG ACTIVITY AT 12 W DURING (A) UPRIGHT AND (B) RECUMBENT CYCLING FOR RECTUS FEMORIS (BLACK), BICEPS FEMORIS (RED), MEDIAL GLUTEUS (BLUE) AND LATERAL GASTROCNEMIUS (GREY).

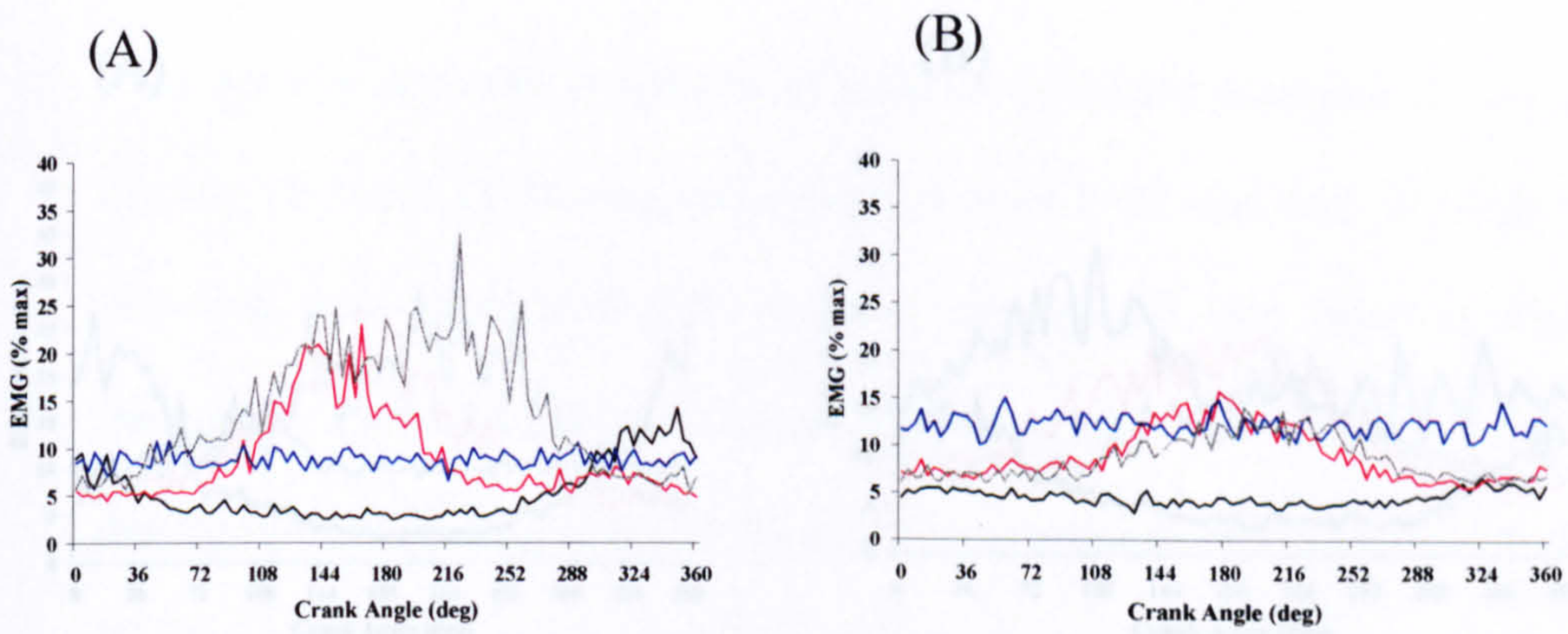

FIG. 7.3: MEAN EMG ACTIVITY AT 25 W DURING (A) UPRIGHT AND (B) RECUMBENT CYCLING FOR RECTUS FEMORIS (BLACK), BICEPS FEMORIS (RED), MEDIAL GLUTEUS (BLUE) AND LATERAL GASTROCNEMIUS (GREY).

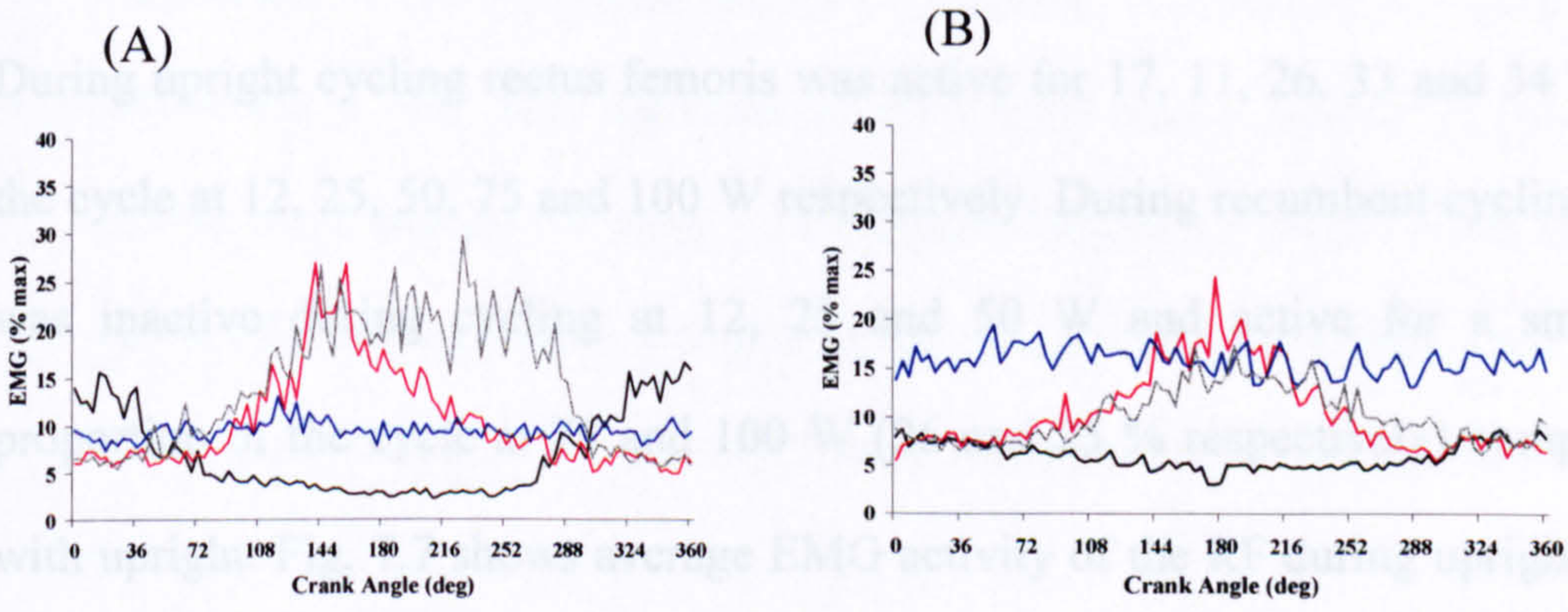

FIG. 7.4: MEAN EMG ACTIVITY AT 50 W DURING (A) UPRIGHT AND (B) RECUMBENT CYCLING FOR RECTUS FEMORIS (BLACK), BICEPS FEMORIS (RED), MEDIAL GLUTEUS (BLUE) AND LATERAL GASTROCNEMIUS (GREY).

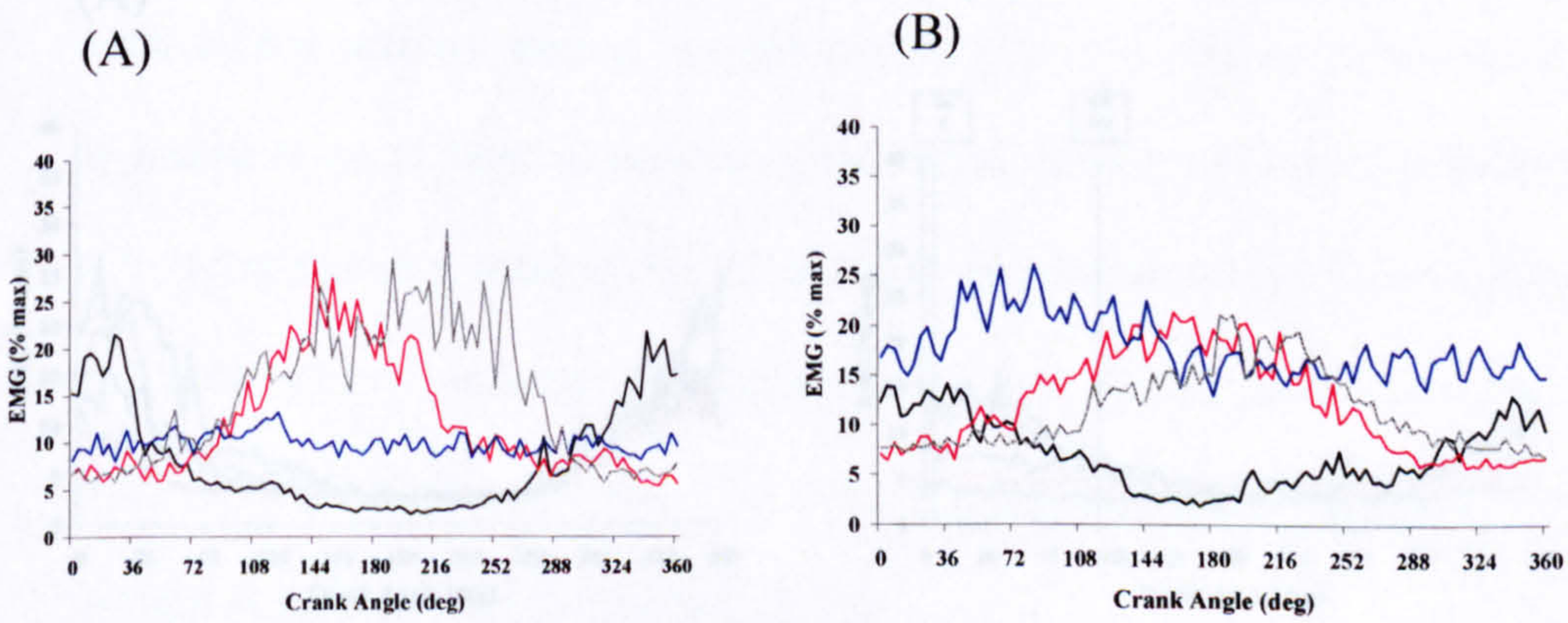

FIG. 7.5: MEAN EMG ACTIVITY AT 75 W DURING (A) UPRIGHT AND (B) RECUMBENT CYCLING FOR RECTUS FEMORIS (BLACK), BICEPS FEMORIS (RED), MEDIAL GLUTEUS (BLUE) AND LATERAL GASTROCNEMIUS (GREY).

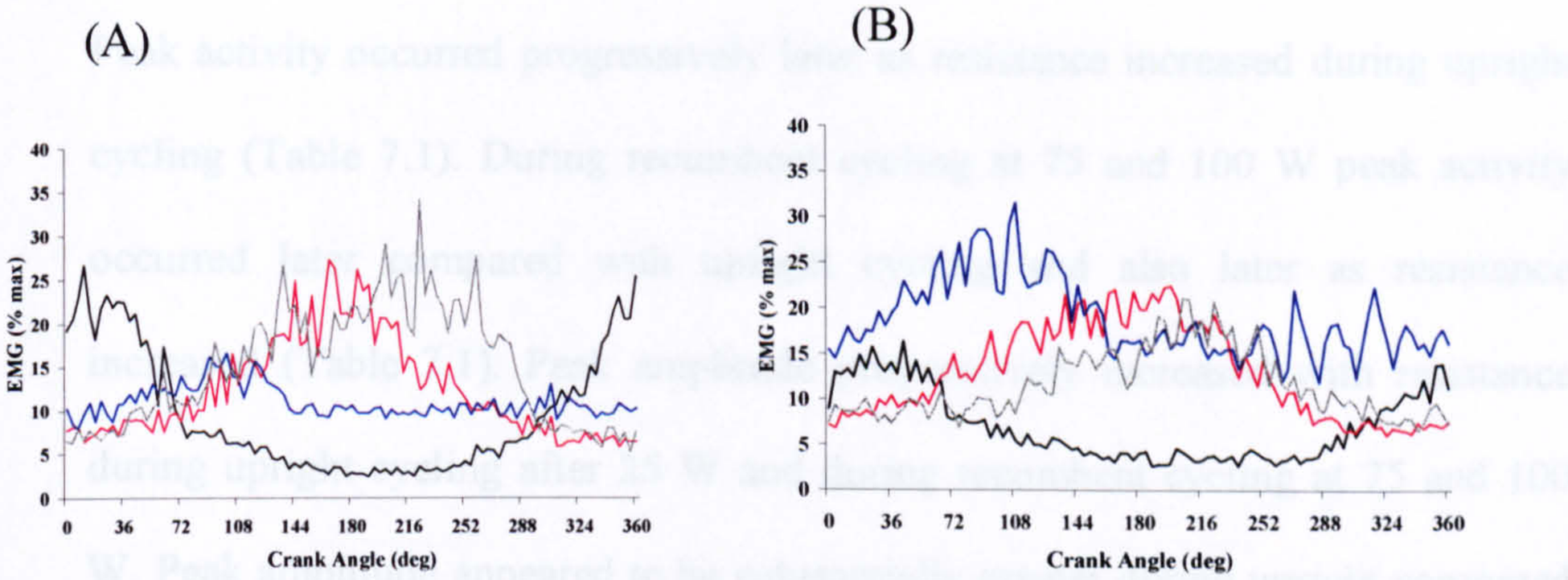

FIG. 7.6: MEAN EMG ACTIVITY AT 100 W DURING (A) UPRIGHT AND (B) RECUMBENT CYCLING FOR RECTUS FEMORIS (BLACK), BICEPS FEMORIS (RED), MEDIAL GLUTEUS (BLUE) AND LATERAL GASTROCNEMIUS (GREY).

During upright cycling rectus femoris was active for 17, 11, 26, 33 and 34 % of the cycle at 12, 25, 50, 75 and 100 W respectively. During recumbent cycling RF was inactive during cycling at 12, 25 and 50 W and active for a smaller proportion of the cycle at 75 and 100 W (26 and 25 % respectively) compared with upright. Fig. 7.7 shows average EMG activity of the RF during upright and recumbent cycling.

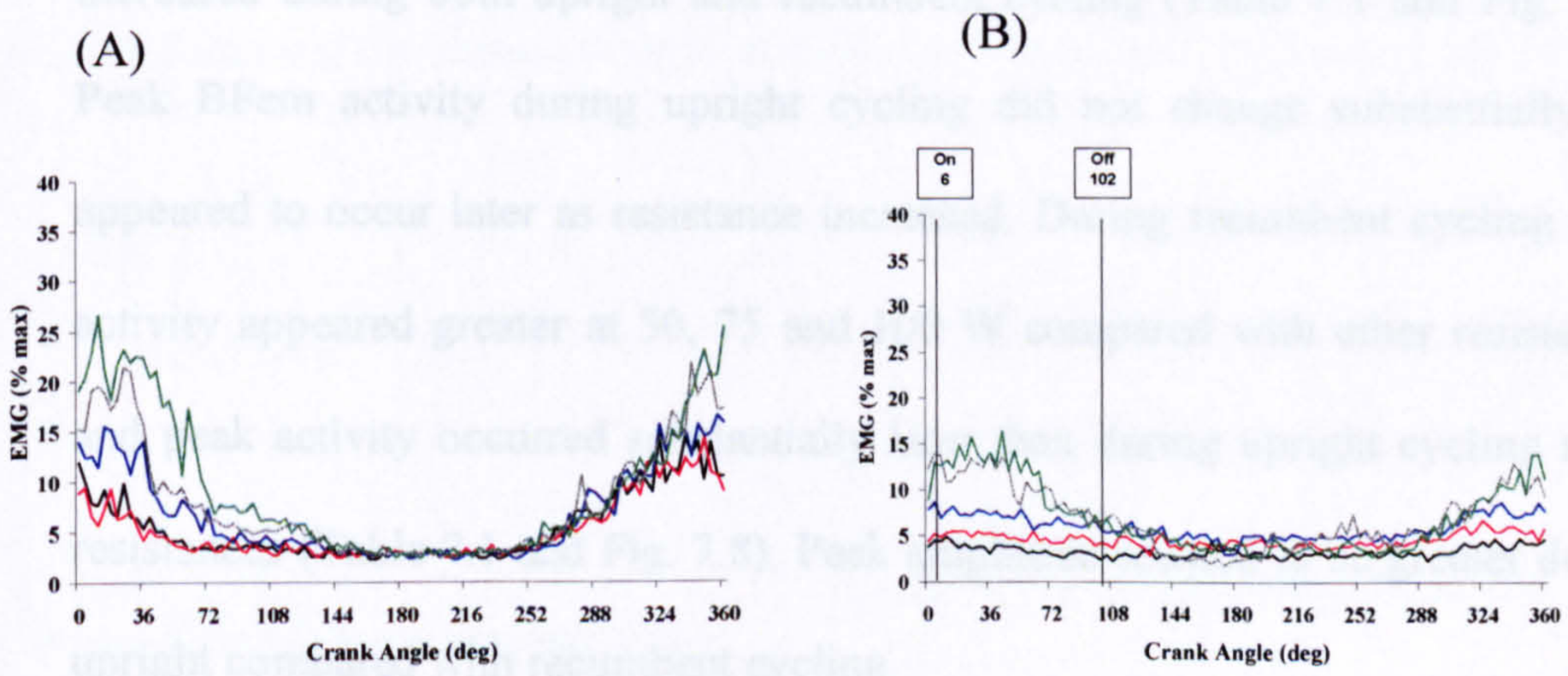

FIG. 7.7: EMG ACTIVITY OF RECTUS FEMORIS DURING (A) UPRIGHT AND (B) RECUMBENT CYCLING AT 12 (BLACK), 25 (RED), 50 (BLUE), 75 (GREY) AND 100 (GREEN) W. VERTICAL LINES INDICATE ONSET AND OFFSET OF QUADRICEPS ACTIVITY USED DURING FES CYCLING.

Peak activity occurred progressively later as resistance increased during upright cycling (Table 7.1). During recumbent cycling at 75 and 100 W peak activity occurred later compared with upright cycling and also later as resistance increased (Table 7.1). Peak amplitude progressively increased with resistance during upright cycling after 25 W and during recumbent cycling at 75 and 100 W. Peak amplitude appeared to be substantially greater during upright compared with recumbent cycling at 75 and 100 W (Table 7.1 and Fig. 7.7).

Stimulation of the quadriceps during FES cycling switches on just after TDC (4°), which is later than the onset of RF activity during voluntary recumbent cycling at 75 and 100 W (Fig. 7.7b). Quadriceps activity switched off at 102° during FES cycling, which is similar to RF during voluntary cycling. There were clear on/off ramps in RF activity during voluntary recumbent cycling, which does not occur during FES cycling.

Duration of biceps femoris activity increased considerably as resistance increased during both upright and recumbent cycling (Table 7.1 and Fig. 7.8). Peak BFem activity during upright cycling did not change substantially but appeared to occur later as resistance increased. During recumbent cycling peak activity appeared greater at 50, 75 and 100 W compared with other resistances and peak activity occurred substantially later than during upright cycling at all resistances (Table 7.1 and Fig. 7.8). Peak amplitude seemed to be greater during upright compared with recumbent cycling.

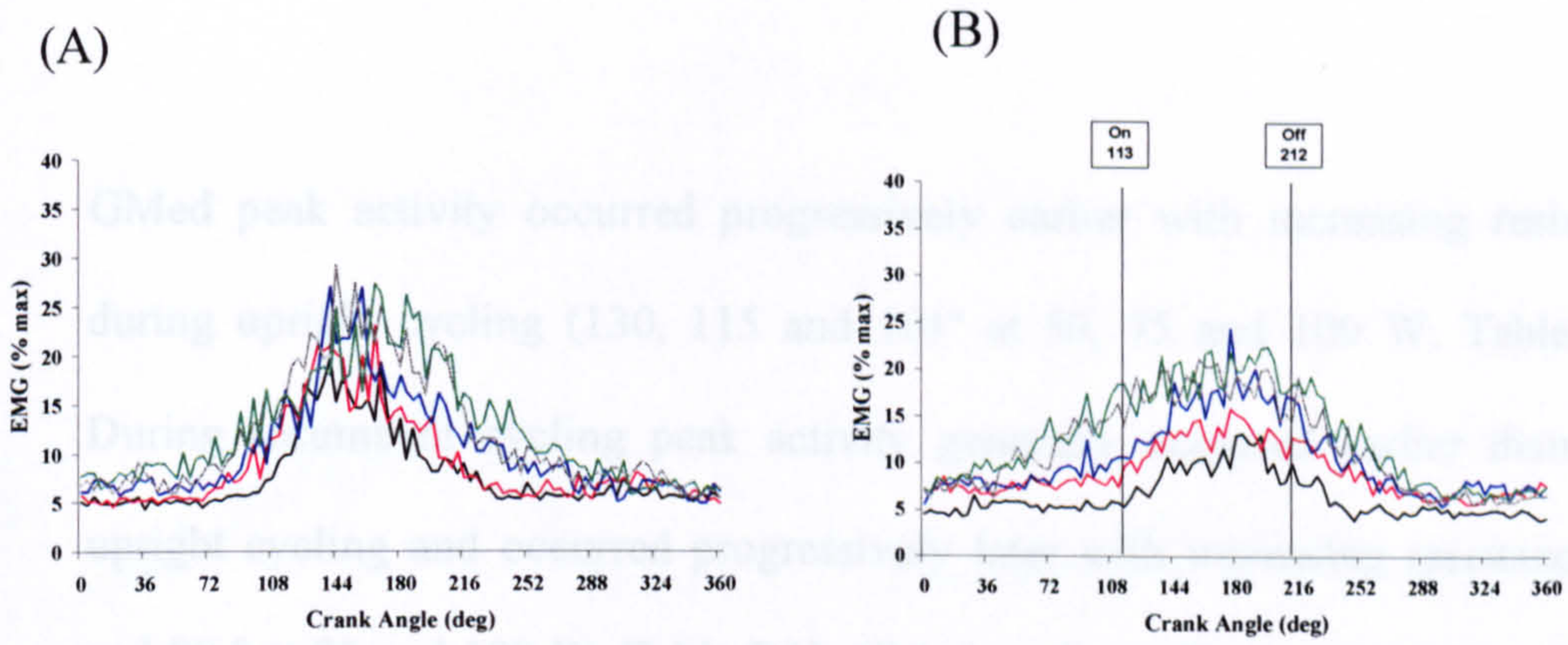

FIG. 7.8: EMG ACTIVITY OF BICEPS FEMORIS DURING (A) UPRIGHT AND (B) RECUMBENT CYCLING AT 12 (BLACK), 25 (RED), 50 (BLUE), 75 (GREY) AND 100 (GREEN) W. VERTICAL LINES INDICATE ONSET AND OFFSET OF HAMSTRINGS ACTIVITY USED DURING FES CYCLING.

Hamstrings activity during FES cycling appeared to be substantially shorter compared with voluntary recumbent cycling (Fig. 7.8b). Voluntary activity started earlier and terminated later compared with ES activation at all intensities and ramped on and off.

Gluteus medius was inactive at 12 and 25 W during both upright and recumbent cycling and also at 50 W during recumbent cycling (Table 7.1 and Fig. 7.9). At other resistances, duration of GMed activity increased with increasing resistance.

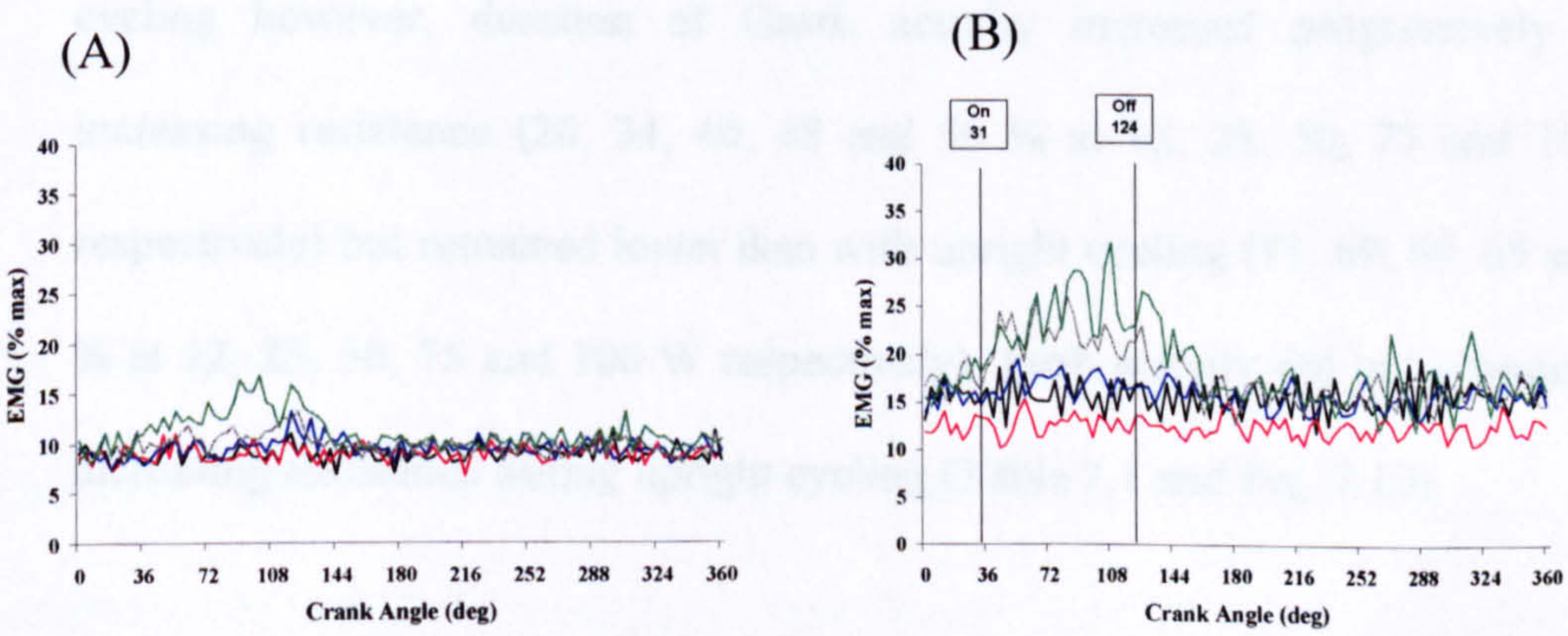

FIG. 7.9: EMG ACTIVITY OF MEDIAL GLUTEUS DURING (A) UPRIGHT AND (B) RECUMBENT CYCLING AT 12 (BLACK), 25 (RED), 50 (BLUE), 75 (GREY) AND 100 (GREEN) W. VERTICAL LINES INDICATE ONSET AND OFFSET OF GLUTEAL ACTIVITY USED DURING FES CYCLING.

GMed peak activity occurred progressively earlier with increasing resistance during upright cycling (130, 115 and 101° at 50, 75 and 100 W, Table 7.1). During recumbent cycling peak activity generally occurred earlier than with upright cycling and occurred progressively later with increasing resistance (65 and 90 ° at 75 and 100 W, Table 7.1). GMed peak amplitude appeared greater at 100 compared with 75 W during both upright and recumbent cycling (Table 7.1). In contrast with other muscle groups, relative peak amplitude of the GMed was considerably higher during recumbent than upright cycling.

Onset of gluteal activity during FES cycling (31°) was similar compared with Gmed activity during voluntary recumbent cycling at 75 and 100 W. However, Gluteal ES activation terminated earlier than Gmed voluntary activation (Fig 7.9b). Gmed voluntary activation ramped on and off, which does not occur during FES gluteal activation.

Duration of lateral gastrocnemius activity did not change substantially with increasing resistance during upright cycling (Fig. 7.10a). During recumbent cycling however, duration of GastL activity increased progressively with increasing resistance (20, 34, 40, 48 and 56 % at 12, 25, 50, 75 and 100 W respectively) but remained lower than with upright cycling (71, 69, 69, 65 and 69 % at 12, 25, 50, 75 and 100 W respectively). Peak activity did not change with increasing resistance during upright cycling (Table 7.1 and Fig. 7.10).

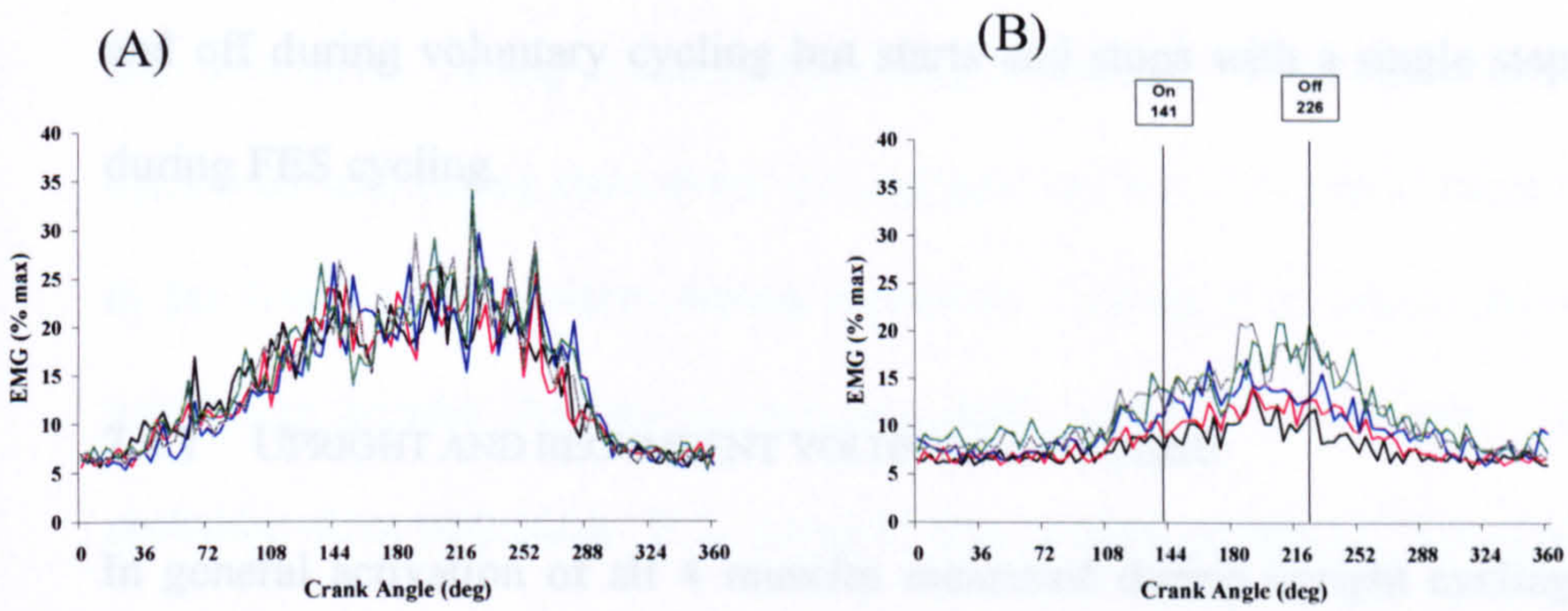

FIG. 7.10: EMG ACTIVITY OF LATERAL GASTROCNEMIUS DURING (A) UPRIGHT AND (B) RECUMBENT CYCLING AT 12 (BLACK), 25 (RED), 50 (BLUE), 75 (GREY) AND 100 (GREEN) W. VERTICAL LINES INDICATE ONSET AND OFFSET OF GASTROCNEMIUS ACTIVITY USED DURING FES CYCLING.

Peak GastL amplitude during recumbent cycling however increased progressively with resistance until 75 W and did not change thereafter (Table 7.1 and Fig. 7.10b). At all intensities, relative peak amplitude seemed greater during upright compared with recumbent cycling.

Gastrocnemius activity during FES cycling switched on slightly later and terminated substantially earlier compared with GastL voluntary activity during recumbent cycling (Fig. 7.10b). GastL voluntary activity showed on and off ramping activity, which does not occur during FES cycling.

7.4 DISCUSSION

The main finding of this study was that RF, BFem and GastL made substantially smaller contributions to recumbent than upright cycling at 5–20 % maximum (12-50 W). Additionally, electrical stimulation timing during FES cycling appeared to be shorter for all muscle groups than occurred during voluntary

recumbent cycling and the shape of activity differed substantially i.e. ramped on and off during voluntary cycling but starts and stops with a single step change during FES cycling.

7.4.1 UPRIGHT AND RECUMBENT VOLUNTARY CYCLING

In general activation of all 4 muscles measured during upright cycling was in agreement with previous studies (Carlsöö & Molbech, 1966; Jorge & Hull, 1986 Clarys et al., 1988; Ericson, 1988; Ryan & Gregor, 1992).

At low intensities, overall activation of the muscles measured appeared to be less during recumbent than upright cycling. In particular, activation of RF and GMed were absent during recumbent cycling at low cycling intensities (<75 W or 30 % maximum). RF is a major knee extensor and therefore it was expected it would be activated, at least in part, at low intensities. This suggests that alternative muscles were contributing more to recumbent cycling. Presumably the different leg orientation in recumbent cycling resulted in altered contribution of gravity to the cycling motion, which might have caused altered activation patterns.

Since RF is a biarticular muscle, it is very likely that other muscle groups of the quadriceps (VL and VM) were driving knee extension during recumbent cycling. Similarly BFem activity was lower during recumbent than upright cycling indicating that other muscle groups assisted knee flexion (ST and SM) and hip extension (GMax) (Jorge & Hull, 1986; Ericson et al., 1985; Ericson 1988; Ryan & Gregor, 1992) during recumbent cycling. Alternatively, activity in the muscles of the ankle and foot might have been sufficient to produce the required power

output (Houtz & Fischer, 1959, Ericson et al., 1985). Indeed, GastL was active at low intensities during recumbent cycling. However, GastL also appeared to contribute less during recumbent than upright cycling. This might have been due to the fixed ankle position during recumbent cycling (i.e. due to the orthoses) preventing plantar flexion during the BDC phase where GastL activity is probably most important. It is possible that muscle groups of the contralateral lower limb made a greater contribution to ipsilateral knee extension for example tibialis anterior might be used to dorsiflex the ankle assisting ipsilateral knee flexion and thus contralateral knee extension.

At higher intensities (75 and 100 W or 30 and 41 % maximum) GMed made a substantially greater contribution to recumbent than upright cycling, whereas other muscle groups appeared to contribute less. The more horizontal body configuration and presence of a backrest in recumbent cycling presumably allowed people to use their body mass against the backrest in order to assist hip and knee extension during the driving phase. It has been suggested that GMed contributes to prevent knee abduction or external rotation of the femur (Ericson et al., 1985; Ericson, 1988). Greater GMed contribution might therefore be due to the more reclined position resulting in a tendency for knee abduction and lateral rotation of the thigh, which does not exist during upright cycling.

7.4.1.1 Effect of changing load

In agreement with previous studies (Houtz & Fischer, 1959; Ericson et al., 1985; Jorge & Hull, 1986; Clarys et al., 1988) EMG amplitude increased with increasing load during both upright and recumbent cycling, except GastL where

amplitude was unchanged during upright cycling. Similarly, duration of activity increased with increasing load during both upright and recumbent cycling for all muscles except GastL and RF remained unchanged during upright and recumbent cycling, respectively. Presumably a greater number of motor units were recruited to attain higher power outputs as load increased resulting in increased peak EMG amplitude. Duration of activity probably tended to increase to allow a smooth synchronised transition between muscle groups (i.e. to maintain momentum).

Changes in the angle of peak amplitude for each muscle were variable. During upright cycling peak amplitude of RF and BFem occurred progressively later, GMed occurred progressively earlier and GastL remained unchanged with increasing load. During recumbent cycling peak amplitude of BFem occurred progressively earlier, GMed occurred later and RF and GastL remained unchanged. These changes appeared to relate to changes in duration of activity i.e. whether activation started earlier or switched off later.

7.4.2 VOLUNTARY AND FES RECUMBENT CYCLING

Current FES cycling stimulation protocols are largely based on voluntary upright cycling. As shown in section 7.4.1, there are clear differences between upright and recumbent cycling in terms of muscle activation patterns. Consequently the differences in activation patterns between FES and voluntary recumbent cycling might result in reduced performance during FES cycling.

The onset and offset of quadriceps activity occurs at 6 and 102° respectively during FES cycling. RF during voluntary recumbent cycling was inactive at 5-20

% maximum (12-50 W) and at higher resistances appeared to end much earlier in the power phase (~65-68°). Current FES cycling patterns where RF is active through out the power phase might be inducing hip flexion, which counteracts the actions of knee extensors (VL and VM). VL and VM have been shown to remain active later in the power phase compared with RF during voluntary upright cycling (Jorge & Hull, 1986). It might therefore be more beneficial to activate VL and VM alone at low workloads and only activate RF during the late recovery-early power phase at higher resistances during FES cycling.

It should be noted that VL and VM are probably the most important muscles for knee extension during cycling because their action is over the knee joint only, whereas RF is a biarticular muscle. Therefore it is very important that future work is carried out to determine the actions of these muscle groups during voluntary recumbent cycling. These results could then be applied to FES cycling by activating VL and VM separately to RF, probably later in the power phase (Jorge & Hull, 1986), which might substantially contribute to the torque created during the power phase and thus overall power output.

The hamstrings were activated at 113-212° during FES cycling, which closely resembles BFem peak activity during voluntary upright cycling. Peak activity occurred later during recumbent cycling and was less pronounced. It is possible that delaying and reducing BFem activity would be more beneficial during recumbent cycling in order to prevent excessive hip extension at the start of the recovery phase. Separate activation of the BFem and the other two hamstring muscles might further improve performance during FES cycling.

At low cycling intensities (5-20 % maximum) gluteal muscles were inactive during recumbent cycling indicating that these muscles are not required. The activation of these muscles during FES cycling at low intensities might therefore be reducing cycling efficiency. However, activation of gluteals is probably beneficial for SCI people because maximum seating pressures around the ischeal tuberosities have been shown to be significantly reduced and redistributed to surrounding areas during ES in SCI people (Levine et al., 1989; Ferguson et al., 1992; Bogie et al., 2000). Thus it is probably more beneficial to activate gluteals, even at low intensities, to maintain muscle bulk and reduce pressure sore risk during FES cycling.

The activation of gluteal muscles during FES cycling appear to closely match activity during voluntary recumbent cycling at high intensities (Fig. 7.9) except activity continued for slightly longer into the power phase during voluntary cycling. Therefore, extending gluteal activation during FES cycling might be beneficial.

Gastrocnemius activity occurred from 141-226° during FES cycling. This is approximately the same as EMG activity recorded at low resistances during voluntary recumbent cycling (Table 7.1). During upright cycling, GastM has been shown to carry out a slightly different role, activating earlier during the power phase. Therefore separate GastL and GastM activation might be beneficial. However, further work is required to investigate GastM activity during recumbent cycling. In addition, use of orthoses that allow plantarflexion at the ankle might improve the contribution of gastrocnemius activation to PO.

In general muscle activation during FES cycling involves a constant stimulation intensity for each muscle group. It is clear from Figs. 7.7-7.10 that all muscle groups are naturally recruited and de-recruited over a ramp with shorter periods of peak activation. Since certain two joint muscles might be working against the forward cycling motion at particular times during the cycle, it is possible that ramping on and off the stimulation would allow a more fluent transition between the working muscle groups and the activity to generate more power. Fig. 7.11 shows (a) the current FES cycling set up and (b) a possible new set up based on data collected in the present study from AB people cycling voluntarily on a recumbent trike at 100 W.

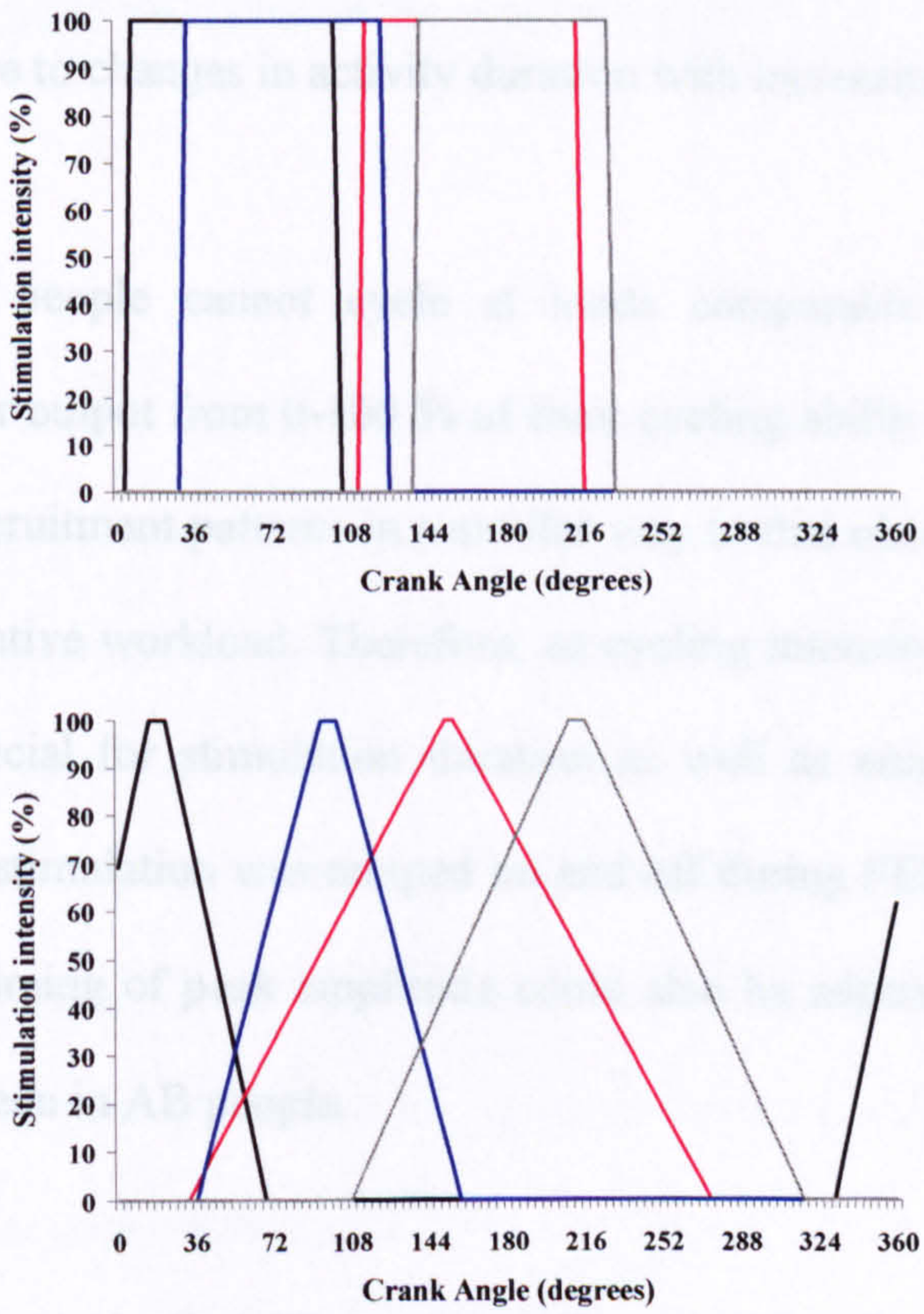

FIG. 7.11: CURRENT FES CYCLING SET UP (A) AND POSSIBLE NEW SET UP BASED ON DATA COLLECTED IN THE PRESENT STUDY ON AB PEOPLE DURING VOLUNTARY RECUMBENT CYCLING (B). (BLACK = QUADRICEPS, RED = HAMSTRINGS, BLUE = GLUTEALS AND GREY = GASTROCNEMIUS). (CRANK ANGLE 0° = LEFT TDC).

7.4.2.1 Effect of changing load

During FES cycling SCI people are in control of the stimulation intensity by use of a throttle (see Chapter 2) in order to increase activation of all muscle groups. However, duration of stimulation is unchanged and the muscles are switched on and off in one step and thus peak amplitude is also unchanged. As mentioned previously, duration of activity gradually increases with increasing cycling intensity for all muscles measured (except RF) during voluntary recumbent cycling. This results in the duration of activity being substantially longer for all muscle groups (except RF) during voluntary compared with FES cycling at ~20-41 % maximum. Furthermore there is a gradual recruitment and de-recruitment of each muscle during voluntary cycling resulting in peak amplitudes that appear to adjust relative to changes in activity duration with increasing load.

Although SCI people cannot cycle at loads comparable with AB people, adjusting power output from 0-100 % of their cycling ability would presumably alter muscle recruitment patterns in a similar way to that observed in AB people at the same relative workload. Therefore, as cycling intensity increases it might be more beneficial for stimulation duration as well as amplitude to increase. Furthermore if stimulation was ramped on and off during FES cycling as shown in Fig. 11(b), timing of peak amplitude could also be adjusted to more closely match what is seen in AB people.

Finally, it is noteworthy that AB people cycling at similar absolute power outputs to those carried out by SCI people (~12 W) exhibit little or no observable contraction of muscle groups. During FES cycling in SCI people however, very

strong contractions of muscle groups can be observed. This suggests that during voluntary cycling at low intensities precise activation of motor units within a number of different muscle groups create the cycling motion. Such activation would be very difficult to replicate using FES.

7.4.3 LIMITATIONS

This study was limited because it was not possible to obtain true maximum values for both EMG activity and cycling ability in the subjects tested. This was because the recumbent trike can be pedaled at a maximum intensity of 250 W only. Additionally, Matlab scripts to provide a statistical analysis of the data were not available to the author at the time of the study.

7.5 CONCLUSIONS

This study showed that relative peak amplitudes and duration of activity for RF, BFem and GastL seemed to be greater during upright compared with recumbent cycling at any given power output. This suggests that additional muscle groups make a greater contribution to recumbent cycling. During recumbent cycling at low resistance muscles of the foot and ankle might play a more important role. At higher resistances, GMed and possibly VI, VL and VM appear to be more important in the power phase than RF, possibly because of the role of RF in hip flexion as well as knee extension.

Voluntary recumbent cycling differs from FES mainly in two ways: i) RF and GMed activation are absent at low intensities and ii) duration of muscle activity

is substantially longer at ~20-41 % maximum (50-100 W). Altering FES cycling stimulation patterns to more closely resemble those seen in AB people would presumably improve cycling efficiency and possibly power output. Altering stimulation to allow a ramp on and off might further improve FES cycle performance. Future work should be directed towards investigating the actions of alternative muscle groups of the quadriceps, hamstring, gluteal and gastrocnemius muscles to determine their relative contributions and whether these differ from the muscles measured in the present study.

The final chapter summarises all preceding chapters highlighting limitations in the work presented as well as providing a rationale for future directions in the field of FES cycling.

Chapter 8 Summary and conclusions

8.1 Introduction

This thesis investigated i) the effects of a one year functional electrical stimulation (FES) cycling training programme on muscular properties, cardiovascular fitness and pressure sore susceptibility in people with spinal cord injury (SCI), ii) power output and the associated metabolic responses to FES cycling following training and iii) muscle activation patterns in able bodied (AB) people during both upright and recumbent voluntary cycling. It was the overall aim of this project to identify the potential health benefits of a long term FES cycling programme for people with SCI and to improve the current FES cycling set up.

A complete SCI results in complete sensory and motor paralysis below the lesion level. Consequently these individuals experience substantial reductions in lower limb muscle size (e.g. Castro et al., 1999a,b; Gerrits et al., 1999; Modlesky et al., 2003; Elder et al., 2004) and strength (Rochester et al., 1995a; Gerrits et al., 1999) as well as a fibre-type transformation to predominantly or exclusively type II fatigable fibres (e.g. Chilibeck et al., 1999; Rochester et al., 1995b). Reduced muscle size results in increased pressure sore susceptibility due to the reduced padding surrounding bony prominences. Reduced blood flow to the lower limbs also occurs, which might further increase pressure sore susceptibility by reducing tissue oxygenation. A substantial loss in cardiopulmonary fitness also occurs following chronic SCI (Glaser, 1986) due to an inability to exercise the large muscle mass of the lower limbs. This results in an increased susceptibility to

cardiovascular diseases including heart disease and stroke as well as type II diabetes.

FES cycling, which involves electrical stimulation of 4 muscle groups in the lower limbs to produce a cycling motion (Perkins et al., 2001), provides the potential to reverse some of the detrimental effects of chronic SCI. Previous studies investigating the potential benefits of FES cycling have used short term programmes involving training for 30 minutes, three times per week. There has been no study to date reporting the potential health benefits of an intense long term FES cycle training programme. In AB people, greater health benefits are known to occur in response to more vigorous and prolonged activity (Åstrand, 1997). Therefore the present study recruited people with complete SCI to carry out a one year FES cycle programme, training one hour per day, 5 days per week. It was hypothesised that such a training regimen would result in more pronounced health benefits in SCI people than has been reported previously.

Previous studies that have investigated FES cycling programmes have reported low power outputs (<55 W) than normal (Petrofsky & Stacy, 1992; Goss et al., 1992; Hooker et al., 1992; Mohr et al., 1997). Therefore an additional aim of this project was to investigate reasons for these low power outputs in order to allow FES cycling to become a more realistic exercise modality for SCI people.

8.2 THE EFFECTS OF A ONE YEAR FES CYCLE TRAINING PROGRAMME

8.2.1 POWER OUTPUT

As a result of a one year FES cycle training programme power output increased from 5.8 W at baseline to 7.4, 14.0, 16.9 and 19.4 at 3, 6, 9 and 12 months respectively. These power outputs were comparable with those previously reported from both short (Goss et al., 1992; Hooker et al., 1992) and long (Mohr et al., 1997) term FES cycle training programmes incorporating training at a lower frequency (three times per week). This suggests that training at a higher frequency (5 times per week) does not result in substantial improvements in power output. It is possible that FES cycling resulted in neuromuscular damage and inadequate recovery time reduced power output during subsequent sessions. Indeed, SCI people have been reported to have a greater relative area of stimulated quadriceps muscle injured three days after ES induced isometric exercise than AB people (Bickel et al., 2004a). This resulted in reductions in peak torque of 25 and 2 % for SCI and AB people, respectively. The wide day-to-day variations in training ability reported by subjects in the present study might also be due to muscle damage. It has been suggested that muscle damage is the basis to hypertrophy because the body overcompensates for microtrauma to the muscle fibres. However there is little evidence to support this view. Similar variations in cycling ability have been reported previously (Ragnarrson et al., 1988; Mohr et al., 1997; Gerrits et al., 2000).

8.2.2 MUSCLE CONTRACTILE PROPERTIES

Both muscle size and strength improved significantly as a result of training. However, the subjects remained significantly weaker than AB people despite similar quadriceps muscle thickness. It is likely that the ES did not activate all muscle fibres of the quadriceps during both training and tests of maximal isometric torque in SCI people. Furthermore the unnatural recruitment of muscle fibres during ES may have caused SCI people to remain significantly weaker than AB people because the entire muscle group might not be activated during ES and synchronous firing of motor units causes rapid fatigue. This is particularly valid considering that AB people were tested for maximal voluntary torque and are able to exercise voluntarily (although these subjects were untrained).

The relative improvements in strength of the quadriceps muscle as a result of the training were substantially greater than the improvements in peak power output assessed during an incremental exercise test (Fig. 8.1).

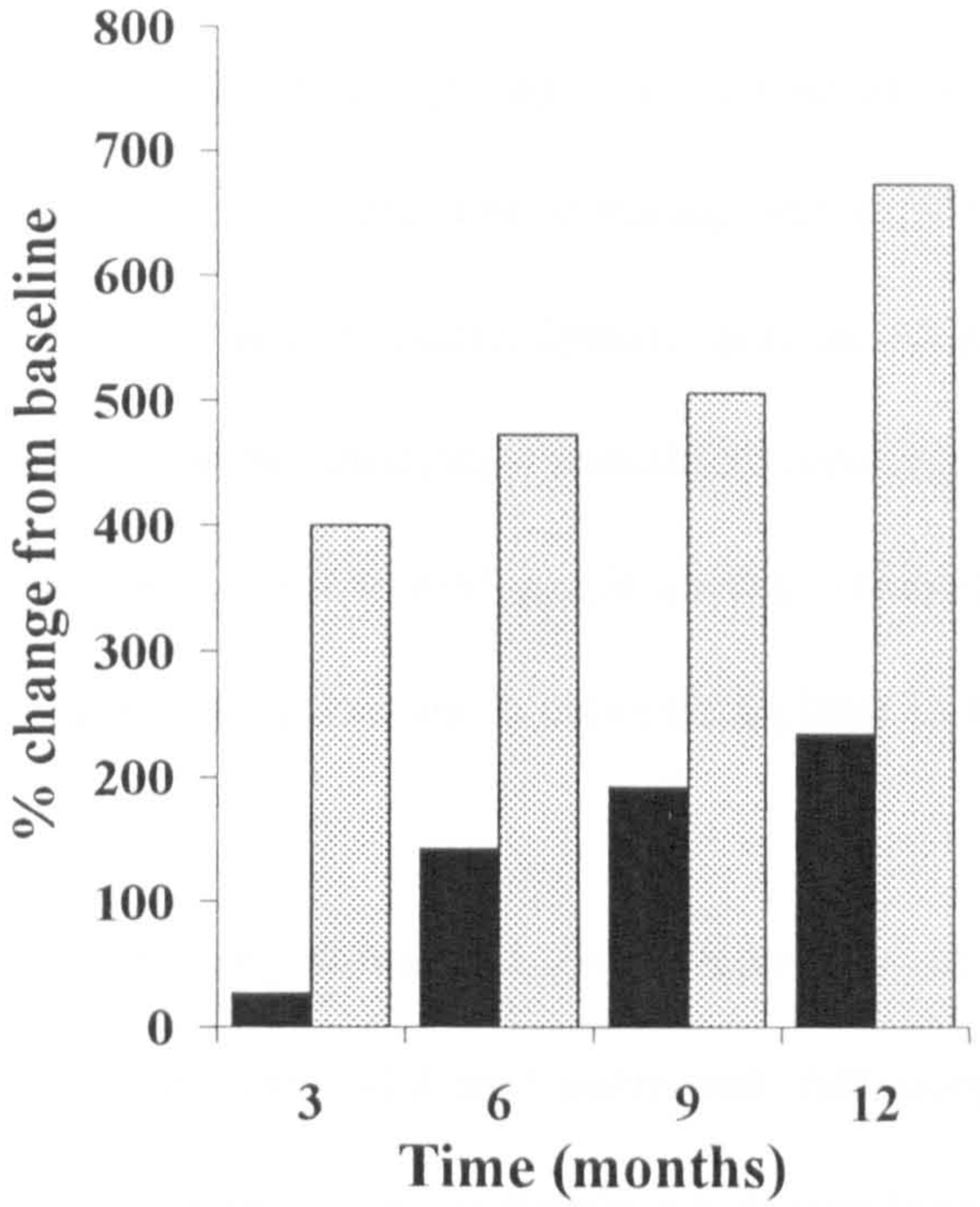

FIG. 8.1: RELATIVE CHANGES IN POWER OUTPUT (BLACK BARS) AND QUADRICEPS FORCE (GREY BARS) FOLLOWING 3, 6, 9 AND 12 MONTHS FES CYCLE TRAINING COMPARED WITH BASELINE.

This suggests that the additional muscle strength following an FES training programme is being dissipated during FES cycling resulting in lower power outputs than expected. This is in agreement with the low efficiency levels observed in SCI people during FES cycling both pre (6.1 ± 1.1 %) and post (12.2 ± 2.4 %) training.

A number of factors might contribute to this inefficiency. The constant high stimulation frequency (50 Hz) is unnatural and so a build up of intramuscular metabolites might limit the contractile force that can be produced by each motor unit. This might be further exaggerated by synchronous stimulation of motor units throughout the training. AB people are able to offset fatigue to some extent by asynchronous recruitment of muscle fibres and also by reducing firing

frequency as the muscle fatigues and becomes slower. Additionally, during FES cycling each muscle group is activated as a single functional unit for a given duration in the cycle. For example, the quadriceps are activated to create knee extension, however rectus femoris acts as a hip flexor as well as a knee extensor. Therefore some antagonist muscle actions might be counteracting the production of power, which would result in the observed reduction in efficiency. These points will be discussed in more detail later in this chapter.

Before training some SCI people appeared to have a substantially larger twitch force than other SCI and untrained AB people. It is interesting to note that although there was no correlation between those subjects with a larger twitch size and age, lesion level, time since injury, muscle size and power output measured during an IET, they were the weakest at the start of the study and those that experienced most difficulty in building cycling ability during the initial three months of training. Previous studies have shown some people to maintain a greater proportion of slow twitch fibres than others following SCI (Hartkopp et al., 1999; Gerrits et al., 2003). Therefore the reason for variability in cycling ability at the start of training might be related to variations in fibre type composition. This requires further investigation.

The torque:frequency ratio shifted to the right with training which was probably due to a reduction in activation per impulse and therefore lower relative force at low frequencies. Furthermore, relaxation rate from tetani reduced with training, which might further contribute to this rightward shift. Alternatively, changes in muscle temperature or tendon properties after training may have had an affect.

Fatigue resistance was significantly lower for all SCI people before training compared with AB people. This improved significantly after three months training and continued to improve gradually thereafter. By 12 months SCI people appeared to be more fatigue resistant than untrained AB people. In contrast to the rightward shift of the force frequency relationship, this suggests that a fast to slow fibre type transformation occurred. Improvements in fatigue resistance have been shown to occur irrespective of stimulation frequency. The long duration of training (one hour per session) would also promote fatigue resistance. It is possible that improvements in blood flow or capilliarisation, improved mitochondrial density and increased enzymes associated with oxidative metabolism resulted in improved oxygen delivery and uptake in the muscle and thus improved fatigue resistance.

Overall, the contractile process appeared to be impaired in some SCI people resulting in an exaggerated twitch size. FES training appeared to shift this process to more closely match AB people. Fatigue resistance improved significantly in response to intense FES cycle training, despite the rightward shift in the torque:frequency relationship.

8.2.3 CARDIOVASCULAR ADAPTATIONS

Improvements in peak oxygen uptake ($\dot{V}O_2$) and ventilation (V) were small but significant in response to training. There was no significant change in peak or resting heart rate. It should be noted that SCI people attained very low peak $\dot{V}O_2$,

HR, RPE and RPB before and after training compared with normal values during voluntary $\dot{V}O_2$ max testing. It is therefore likely that the significant improvements in power output due to muscular adaptations allowed individuals to work at a higher $\dot{V}O_2$ after training. Thus peripheral as opposed to central adaptations resulted in the significant improvements in peak $\dot{V}O_2$. This is further supported by the fact that peak VO_2 after FES cycle training remained less than that achieved by SCI people during voluntary arm exercise (~1.2-1.4 $L.min^{-1}$) (Hooker et al., 1992; Mutton et al., 1997). Presumably the low power outputs attained during FES cycling were an inadequate stimulus for central cardiovascular adaptations.

It is possible that stimulating a greater number of muscle groups in the lower limb might induce a greater O_2 demand during exercise, which would place a greater strain on the central cardiovascular system. Alternatively, recruiting muscle groups for a longer period per cycle would elicit a similar effect.

Steady state $\dot{V}O_2$ at a given power output reduced significantly as a result of training however steady state HR remained unchanged. This further supports the suggestion that peripheral as opposed to central adaptations took place. Presumably improvements in muscle strength allowed SCI people to work at similar power outputs using relatively lower levels of stimulation intensity because activation of fewer motor units was required to generate similar power outputs after training. This might therefore explain the lower $\dot{V}O_2$ at a given

power output due to training observed in the present study, which would be unexpected to occur in AB people after training.

Cycling efficiency was low for SCI people before training compared with AB people. Although efficiency tended to improve with training, due presumably to the reduction in steady state $\dot{V}O_2$, this did not attain statistical significance and remained lower than would be expected in AB people. As mentioned previously, some antagonist muscle actions might be occurring during FES cycling, which would utilise O_2 but counteract the production of power. Although this would elicit a higher $\dot{V}O_2$ and heart rate, promoting cardiovascular fitness, it limits the attainable power outputs and therefore the outdoor use of FES cycle systems. Efficiency in AB people is only likely to improve if newly learned, optimal, muscle recruitment patterns become more familiar. Since this process is removed in SCI people, it is understandable that efficiency did not improve to a level comparable with AB people. It is important to understand the natural muscle recruitment patterns in AB people during recumbent cycling in order to improve efficiency for SCI people during FES cycling. .

8.2.4 SUSCEPTIBILITY TO PRESSURE SORES

FES cycling has the potential to reduce pressure sore susceptibility in SCI people mainly in two ways; i) improved blood flow to the lower limbs, which might improve tissue oxygenation and recovery from tissue hypoxia and ii) improved muscle bulk around bony prominences such as the ischeal tuberosities.

Measurements of tissue oxygenation made at the sacrum did not change as a result of the one year training programme. The highly significant improvement in fatigue resistance indicated that oxygen supply or delivery to the working muscle improved as a result of training. This suggests that blood flow to the working muscles or capillarisation surrounding the working muscles improved. It is however unclear whether a similar effect would occur for cutaneous capillarisation. Since thermoregulation is known not to occur below the lesion level, it is unlikely that skin blood flow would have increased during FES training indicating that no stimulus for improved capillarisation existed. However it has been reported that ES of the gluteal muscles in SCI people results in local increases in transcutaneous oxygen pressure (T_CPO_2) (Bogie et al., 2000). In support of this, subjects in the present study reported that there legs felt warmer to touch following training sessions (unpublished observations). This suggests that some stimulus for improved cutaneous blood flow was present.

The T_CPO_2 measurements made in the present study were found to vary day to day by approximately 7% in AB people. The variations observed in SCI people across the one year training programme were similar to AB people. Therefore it is possible that the measurement technique was not sensitive enough to detect any changes that might have taken place due to the training programme. This is in agreement with a previous study investigating T_CPO_2 changes in hemiplegics (Daviet et al., 2004). Tissue reactivity also showed no change in response to a one year training programme. However, the measurement technique used did not induce reactive hyperaemia and therefore it is difficult to understand whether changes had taken place due to the training. Further work is required to design a

reliable test that induces reactive hyperaemia. However it should be noted that this test is time consuming and uncomfortable for SCI people. Therefore if a test were to be designed it should be tested on AB people to ensure its validity and reliability.

Finally, it should be noted that the individuals recruited onto this study were required to have no history of pressure sores in the two years prior to the study. Therefore the individuals selected were probably at a lower risk of developing pressure sores, which reduced the potential for improvement with training.

Peak seating pressures in both a standard (NHS) and each subject's own wheelchair did not significantly change due to the training. However, gluteal size did tend to increase with training. This might not have resulted in changes in seating pressure due to low subject numbers and variations in body composition of SCI people prior to training. Additionally, seating pressure data in the present study should be interpreted with caution due to methodological limitations.

8.3 METABOLIC RESPONSES TO FES CYCLING FOLLOWING A ONE YEAR TRAINING PROGRAMME

8.3.1 PEAK POWER TEST

Ten minutes cycling at 100 % stimulation intensity resulted in a rapid decline in power output followed by a partial recovery. The rapid decline in power output was unexpected because SCI people were highly fatigue resistant after training when assessed using repetitive contractions of the quadriceps muscle. However,

during short bouts of intense exercise in AB people, a similar rapid decline in power output has been noted (Lakomy, 1986; Bogdanis et al., 1995). The partial recovery in power output identified here has not to my knowledge been previously reported in AB people. It is possible that high frequency fatigue (HFF) (Jones, 1981; 1996) resulting in increased extracellular potassium concentration (Bigland-Richie et al., 1979; Jones, 1981) and inactivation of the sodium channels (Ruff et al., 1987) was at least part of the cause of rapid fatigue. The use of high followed by low frequency stimulation has been shown to result in improved force generation (Jones, 1981). This pattern of stimulation would more closely match the natural muscle activation that occurs in AB people and might prevent such a severe decline in power output at the start of exercise. It is also possible that the use of variable frequency trains during low frequency stimulation might generate greater force without inducing severe fatigue (Russ & Binder-MacLeod, 1999; Scott & Binder-MacLeod, 2003).

The partial recovery has been noted previously in SCI people carrying out FES cycling. Theisen et al. (2002) noted in 5 SCI people that a peak power was achieved after 2 minutes (10.7 W), which declined and subsequently recovered to a level slightly lower than the peak power (5.3 and 8.2 W at 6 and 20 minutes, respectively). It is possible that the high level of fatigue resulted in some muscle fibres becoming inactive and allowed the removal of intramuscular metabolites or potassium resulting in a partial recovery in power output. This effect appeared to occur to a greater extent in SCI people who were more fatigue resistant. Alternatively the habituation of spinal reflexes reported to occur during ES

exercise in SCI people (Andrews et al., 1989; Gregorič, 1998; Knikou & Conway, 2002; 2005) might have allowed this partial recovery.

8.3.2 HOME TRAINING SIMULATION

During a simulated home training session after a one year FES cycle training programme, SCI people appeared to work at a $\dot{V}O_2$ that was 97-100 % peak (measured during an IET) for the initial 35 minutes. The RER remained > 1.0 during this time. There are a number of factors that promote anaerobic energy production in SCI people using FES exercise including a high proportion of fast twitch fibre types, synchronous recruitment of motor units and high frequency stimulation. Higher glycolytic and phosphocreatine (PCr) degradation has been identified in fast compared to slow twitch fibres (Greenhaff et al., 1994), therefore a greater accumulation of hydrogen ions is likely to occur in the former resulting in increased muscular acidosis. It has also been shown that PCr restoration is slower in fast twitch fibres (Tesch et al., 1989; Soderland & Hultman 1991). In SCI people, this build up of metabolic by-products would not induce pain, which might explain the high RER and gradual reduction in power output during the initial 35 minutes of exercise. The use of high followed by low frequency stimulation or the use of VFT's as well as asynchronous firing of motor units might contribute to a reduction in the anaerobic contribution to exercise and therefore the gradual reduction in power output.

A partial recovery in power output appeared to coincide with RER falling < 1.0 after 35 minutes exercise. This indicates a greater aerobic contribution to exercise, which might allow additional oxygen to become available for the

removal of metabolic by-products thus resulting in the observed recovery of power output.

8.4 MUSCLE ACTIVATION PATTERNS DURING VOLUNTARY AND FES CYCLING

Duration of activity for all four muscle groups used during FES cycling were substantially shorter than during voluntary cycling in AB people. Additionally muscle activity ramped on and off for all 4 muscle groups during voluntary cycling whereas FES cycling produced full activation for the duration of activity.

Current muscle activation patterns used during FES cycling have been based on literature evaluating muscle activation during upright cycling in AB people. Recumbent cycling differs compared with upright cycling mainly in three ways i) leg orientation, which affects the contribution of gravity to the motion, ii) trunk angle, which affects the muscles that span the hip joint and iii) the extent to which body mass can be used is altered. There has been little work investigating muscle activation patterns during recumbent cycling. Additionally changes in activation patterns due to altering cycling intensity have not been considered during FES cycling.

Results indicated that rectus femoris contributed little or no activity during recumbent cycling $\leq$ 50 W. At higher intensities activity began later in the flexion phase during recumbent compared with upright cycling and cycling intensity did not alter activity duration during recumbent cycling. Cycling intensity appeared to increase duration of biceps femoris activity during both recumbent and upright cycling, but this was more pronounced during recumbent

cycling. Duration of activity was 15, 44 and 65 % at 12, 50 and 100 W respectively during recumbent cycling and comparative values during upright cycling were 18, 40 and 54 %, respectively. There was no detectable gluteus medius activity at low intensities during both upright and recumbent cycling. Gluteus medius activity was similar between upright and recumbent cycling at higher intensities, except duration of activity appeared to increase with increasing intensity during recumbent cycling only. Lateral gastrocmenius activity started substantially later during recumbent compared with upright cycling at all intensities. Duration of activity during upright cycling was unaffected by cycling intensity, whereas during recumbent cycling activity duration progressively increased with increasing cycling intensity. Muscle activity for all 4 muscle groups increased in amplitude with increasing cycling intensity during both recumbent and upright cycling.

Further work is required to determine activity patterns for individual muscles of the quadriceps, hamstrings, gluteal and gastrocnemius muscle groups. Stimulation of these muscle groups individually might prevent antagonistic muscle actions that are likely to result in reduced power output and reduced efficiency during FES cycling.

8.5 LIMITATIONS AND FUTURE RECOMMENDATIONS

8.5.1 METHODOLOGICAL LIMITATIONS

Major limitations of these studies were low subject numbers and that a control SCI group was not used. The majority of studies carried out with SCI people

involve low subject numbers because it is difficult to recruit SCI people, particularly for a study as time consuming as the present one. Furthermore, the funding available for the present study limited subject numbers to 5 per centre. Data from all three centres will be combined for future publications.

Recruitment of control subjects was unrealistic because the demands of the study were large and there were no real benefits for control subjects. The fact that a control group was not used means that we cannot discount the possibility that the adaptations found were greater than the changes that occur with a one year period or than the variability in the techniques used. Nonetheless, adaptations due to SCI reach a steady state 1-2 years after the injury (except for changes in bone density, which can take up to 7 years to reach a steady state). Therefore, assuming there were no major lifestyle changes in the subjects recruited except the addition of FES cycling (as requested at the start of the study), we can be reasonably certain that adaptations were as a result of the training programme.

The present study identified significant improvements in quadriceps muscle size, strength and fatigability as well as peak power, oxygen uptake and ventilation during FES cycling. However, we were unable to provide strong evidence for additional health benefits due mainly to methodological limitations.

8.5.1.1 Fatigability

As demonstrated during a peak power test (Chapter 6) SCI people were highly fatigable during FES cycling after training despite less than normal fatigue when tested by repeated brief isometric contractions of the quadriceps muscle group

(see Chapter 3). The main difference between these tests was the stimulation intensity (100 and 20-25 % for peak power test and isometric fatigability test respectively). In AB people, measurements of contractile properties (as used in the present study, see Chapter 3) are similar at any stimulation intensity that produces > 20 % maximum isometric strength. It is unknown whether this is true for the SCI population. It would be useful to carry out isometric fatigability tests at 100 % stimulation intensity in order to determine whether the severe fatigue observed during FES cycling in the present study (Chapter 6) was due to high relative stimulation intensity or some factor related to the cycling itself.

8.5.1.2 Improvements in cardiovascular fitness

Although a significant increase in $\dot{V}O_2$ max in AB people after training indicates significant improvements in cardiovascular fitness, it should be noted that similar presumptions cannot be taken from the improved $\dot{V}O_2$ peak observed in the present study. Peak HR, $\dot{V}O_2$, RPE and RPB remained very low at the termination of the IET in SCI people both before and after training. This suggests that peripheral as opposed to central limiting factors always caused fatigue. Thus it is likely that peripheral adaptations due to the training allowed SCI people to attain higher peak power and therefore $\dot{V}O_2$ during the IET after training.

It also must be considered that it was not possible to control electrode position through out the training programme and a control group was not used in this study. Therefore it is possible that subjects had learned the optimal electrode

positioning during training, which resulted in a relatively greater muscle mass being stimulated and the observed improvements in peak power and $\dot{V}O_2$.

8.5.1.3 Pressure sore susceptibility

Tissue oxygenation (T_CPO_2) at the sacrum was not significantly different between AB and SCI people before training. Furthermore, baseline T_CPO_2 and recovery from a known load did not change as a result of a one year FES cycle training programme. This was possibly due to day-to-day variations in T_CPO_2 values, because individuals selected for the study were less susceptible to pressure sores than the average SCI population and the technique lacked repeatability.

It is important to identify a measurement technique that discriminates between tissue oxygenation in SCI and AB people. This could then be used to assess any changes that might occur as a result of an FES training programme. It is also recommended that pressure sore prevalence be monitored in a randomly selected group of SCI people before, during and after an FES training programme or in a group of SCI people that regularly carry out FES training in comparison with a group that do not.

Peak power outputs reported in the present study were relatively low after a one year FES training programme (10-37 W), but similar compared with those reported previously (< 55 W) following short term training programmes carried out at a lower intensity (Arnold et al., 1992; Faghri et al., 1992; Goss et al., 1992; Hooker et al., 1992; Petrofsky & Stacy, 1992). This indicates that

increasing the intensity and/or duration of an FES cycle training programme did not result in improved power output. However, a large limiting factor in the present study was skin damage caused by electrodes, which resulted in reduced compliance to the training programme and long breaks within training. Despite this, average compliance to the training programme was 81 % suggesting that the majority of training was carried out, however the impact of long breaks in training (> 3 months for some subjects) is unknown. It is likely that a number of limiting factors contributed to the low power outputs during FES cycling.

8.5.2 POSSIBLE LIMITING FACTORS IN POWER OUTPUT GENERATION

There are a number of factors that might contribute to high levels of fatigue and low power outputs observed during FES cycling in SCI people. These include both physiological (e.g. changes caused by chronic SCI or altered muscle activation by ES) and mechanical (e.g. timing of muscle recruitment) factors.

Kjær et al. (1994) reported that AB people cycling voluntarily and involuntarily (ES with epidural anaesthesia) at similar $\dot{V}O_2$ achieved significantly lower power output during involuntary exercise (~70-120 and 20-40 W during voluntary and involuntary exercise, respectively). This suggests that low power outputs occur due to the unphysiological recruitment of muscle fibres. However compared with AB people, greater levels of fatigue during ES have been noted in people with SCI (Gerrits et al., 2001b; Olive et al., 2003) indicating that adaptations in untrained SCI people (e.g. slow-fast fibre type transformation) contributed to increased fatigue and thus low power outputs. The present study found that muscle size and fatigue resistance during ES improved significantly in SCI

people after training to levels comparable with AB people, however muscle strength and power output remained very low.

It has been reported previously that despite normal $\dot{V}O_2$, HR and Q responses in AB people exercising with ES, mean arterial blood pressure (MAP) does not rise in response to exercise (Kjær et al., 1994). This might have contributed to the low power outputs in SCI people due to an insufficient rise in blood flow to the working muscles. It has however been reported that increased blood flow to exercising muscle does occur in SCI people in response to ES (Levine et al., 1990b, Olive et al., 2003b). This response has been reported to be similar in AB and SCI people during stimulation of the same amount of muscle mass, despite greater fatigue in SCI people (Olive et al., 2003b). Furthermore, increasing blood flow to stimulated muscles (prior to stimulation) has been reported to not affect the rate of decline in force in SCI people (Olive et al., 2004). Improvements in fatigue resistance have also been reported in SCI people in response to ES training in the absence of vascular adaptations (Sabatier et al., 2006). These findings indicate that impaired muscle blood flow during FES exercise is not the main factor limiting power output for people with SCI.

Although oxygen delivery does not appear to be limited in SCI people, it is unclear whether oxygen uptake in the muscle is limited or prevented during FES cycling. It would therefore be useful to monitor oxy- and deoxyheamoglobin patterns in both AB and SCI people during recumbent cycling. This would enhance our understanding of the intramuscular processes during FES cycling.

Comparing FES with voluntary cycling would allow us to determine whether differences in oxygen uptake affects power output.

Overall it is likely that the unnatural recruitment of muscle fibres due to the pattern and timing of ES is a key factor resulting in low power outputs during FES cycling in SCI people.

8.5.3 FUTURE RECOMMENDATIONS FOR IMPROVEMENTS IN POWER OUTPUT

8.5.3.1 Pattern of electrical stimulation

The stimulation frequency utilised in the present study (50 Hz) was selected in order to create high forces and minimise fatigue as has been reported previously (Kebaetse et al., 2002; Eser et al., 2003; Kebaetse & Binder Macleod, 2004). However in AB people the firing frequency of motor units has been shown to reduce with fatigue due to increasing contraction and relaxation times. Furthermore fatigue caused by high frequency stimulation has been shown to partially recover when frequency is reduced, indicating that changes in the neuromuscular junction cause high frequency fatigue (Jones et al., 1979). It is possible that the high frequency stimulation is causing an intramuscular build up of some metabolite e.g. potassium or magnesium or acetylcholine depletion at the neuromuscular junction, which results in severe fatigue and thus reducing stimulation frequency in fatigued muscle might result in a recovery of power output during FES cycling.

It should however be noted that in the present study fatigability was tested by use of repeated brief isometric contractions of the quadriceps muscle group at 40 Hz (Burke et al., 1971). This resulted in less than normal fatigue (see Chapter 3). Since the duty cycle during the Burke fatigue protocol (25%) is similar to that for quadriceps during FES cycling (27%), fatigue caused by a stimulation frequency of 50 Hz might only be a contributing factor to the severe fatigue observed during FES cycling (see Chapter 6). Even so, only a small recovery in power output is likely to substantially improve cycling ability in SCI people because power output is generally very low in fatigued muscle (10-15 W).

Asynchronous stimulation of muscle fibres increases force and create a smoother contraction compared with synchronous activation (Lind & Petrofsky, 1978; Clamann & Schelhorn, 1988). It has also been reported in both animal (Lind & Petrofsky, 1978; Clamann & Schelhorn, 1988) and human (Binder-Macleod et al., 1997a) muscle that this effect does not occur when the muscle fibres of the two motor units were not anatomically intermingled suggesting that this effect occurs due to mechanical coupling of adjacent muscle fibres (Clamann & Schelhorn, 1988). Asynchronous stimulation of an intermingled population of motor fibres has been reported to produce more force than the sum of the forces produced when the individual motor units are activated individually (Clamann & Schelhorn, 1988). Additionally, the force produced by one motor unit has been reported to increase due to the recruitment and derecruitment of an additional motor unit (Clamann & Schelhorn, 1988). Therefore asynchronous stimulation of an intermingled population of motor units should be investigated to determine

whether greater power outputs are produced during FES cycling i.e. by using a number of paired electrodes for each muscle group.

Alternatively the use of variable frequency trains (VFT's) might create higher power outputs without inducing more fatigue (Binder-Macleod et al., 1997b; Russ & Binder-MacLeod, 1999; Bickel et al., 2003; Scott & Binder-MacLeod, 2003) by making use of the catch-like property of human muscle. VFT's have been reported to increase torque-time integral by 18% in AB people (Bickel et al., 2004b). However the equivalent effect in chronic SCI people was found to be just 6 % (Bickel et al., 2004b) due possibly to the higher proportion of fast twitch fibres (Grimby et al., 1976; Martin et al., 1992; Round et al., 1993; Greve et al., 1993; Rochester et al., 1995b; Chilibeck et al., 1999). It is possible that paralysed muscle trained by ES would result in contractile slowing and the use of VFT's would become more effective and this requires further investigation. Doublet frequency trains (DFT's) have also been found to produce greater peak force and force-time integrals than constant frequency trains (CFT's) and VFT's in both fresh and fatigued muscle of AB people. However, the greatest fatigue was also noted due to DFT's (Binder-Macloed & Scott, 2001). VFT's and CFT's have been shown to have similar effects on fatigue in control and paralysed human muscle (Thomas et al., 2003). The optimal stimulation pattern during FES cycling in both trained and untrained SCI people requires further investigation.

Finally, it would be interesting to further investigate EMG activity of the vastus lateralis and medialis as well as the individual muscles of the hamstrings, gluteals and gastrocnemius. This would help to identify whether activating whole

muscle groups results in some antagonistic actions. If this was the case, overall power output and efficiency during FES cycling would be reduced. Closely replicating voluntary activation by stimulating individual muscles might improve power output and efficiency during FES cycling.

8.5.3.2 Timing of electrical stimulation

Electromyography in AB people during voluntary recumbent cycling showed that voluntary muscle activation occurs for a substantially longer duration than used during FES cycling for all 4 muscle groups tested. It is likely that short compared with long duration contractions result in greater energy costs because the cost of switching on and off each muscle group is greater than maintaining the contraction. Indeed, muscle blood flow has been shown to be higher during short compared to long duration dynamic (Ferguson et al., 2001) and isometric (Hogan et al., 2003) contractions. Furthermore, in isolated animal muscle, short duration isometric contractions have been shown to significantly increase muscle oxygen uptake (Hogan et al., 1998), energy cost of contraction and the rate of decline in force (Bergström & Hultman, 1988; Hogan et al., 1998). Short duration contractions were also reported to result in higher intracellular lactate and H^+ concentrations compared with long duration contractions, which might be the cause of the greater fatigue rate in the former (Hogan et al., 1998). Therefore, shorter duration contractions during FES compared with voluntary cycling might result in greater fatigue and at least in part reduced power output.

It would be useful to replicate the shape and duration of voluntary muscle activation and measure the impact on power output and fatigue during FES

cycling. The effects of altering cycling intensity on duration of muscle activation should also be considered.

The duration of contraction during cycling is also affected by cycling cadence. The well-known force:velocity relationship of muscle shows that faster concentric contractions develop less force than slower ones. This means that during lower cadence cycling, each muscle group will spend longer producing a higher torque per revolution. This is unlikely to affect power output because velocity has decreased. Fornusek & Davis (2004) have identified significantly greater torque production throughout 30 minutes of FES cycling at 15 compared with 50 rpm. However, overall power output was significantly higher during cycling at 50 rpm. This suggests that cycling cadence should be adjusted depending on the type of training (strength or power) required by each individual (Fornusek & Davis, 2004).

8.6 Conclusions

This study was the first to investigate the potential health benefits of a long term, intense programme of functional electrical stimulation (FES) cycling for SCI people. It should be noted that training 5 times per week resulted in skin damage in some SCI people. Furthermore this regimen was time consuming and therefore not ideal for all SCI people.

Significant improvements in peak power output occurred in the first three months of training and continued to improve thereafter. Size, strength and fatigue resistance of the quadriceps also increased significantly. Muscle size and fatigue

resistance were comparable with AB people following the one year training programme, however muscle strength remained significantly less, as did power output during FES cycling. The increase in isometric strength was greater than that of power output. A number of physiological and mechanical factors might cause reduced strength and low power outputs during FES cycling, which require further investigation.

Peak oxygen uptake and ventilation improved significantly following training as did steady state $\dot{V}O_2$. This indicates improved aerobic capacity, probably due to peripheral adaptations. Higher power outputs during FES cycling might bring about central adaptations which would be of greater benefit for reducing susceptibility to cardiovascular disease in SCI people. Efficiency during cycling improved as a result of the training, but the individual changes were variable and did not attain statistical significance.

There have been few studies investigating tissue oxygenation changes in response to FES training programmes. In the present study T_CPO_2 at the sacrum did not change in response to FES training in SCI people. Recovery time from hypoxia induced by a given load also did not change. This was probably due to methodological limitations and large day-to-day variations in tissue oxygenation. Peak seating pressures did not reduce despite a tendency for improvements in gluteal muscle size. This was probably due to low subject numbers, variability in body composition and methodological limitations. Further work is required to assess the effects of FES training on pressure sore susceptibility in SCI people.

The major limitations of this study were low subject numbers and that a control group was not used.

FES cycling for SCI people is a rapidly growing area of interest. Low power outputs and high levels of fatigue during FES cycling limit its use, for example people are unable to cycle on anything other than a smooth flat surface therefore limiting the outdoor use of this type of exercise. Additionally, improvements in central cardiovascular fitness are limited by low power outputs. Further investigation is required to identify physiological mechanisms and optimal stimulation patterns to improve power outputs attainable.

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Appendix 1

INFORMATION SHEET FOR VOLUNTEERS

Brief Title of Project
The development of Functional Electrical Stimulation (FES) systems for tricycling: improving health after spinal cord injury.

Explanation
We would like to ask you to participate in a research project. Please read the following information carefully before deciding. If necessary, go away and discuss this with someone else to help you make up your mind. You are under no obligation to take part in the study and may decline without giving reason, without incurring displeasure or penalty and without affecting your future medical care. You are free to withdraw at any time without explanation.

Complications involved with Spinal Cord Injury (SCI) including loss of muscle mass in the lower limbs, bone thinning, incidence of pressure sores and a loss of cardiopulmonary fitness all contribute to a reduced quality of life for persons with paraplegia. It is possible that Functional Electrical Stimulation (FES) cycling has the potential to provide important health-related benefits contributing to an improved quality of life. Such benefits include increased muscle mass, blood flow and bone density in the lower limbs, improvements in cardiopulmonary fitness as well as improvements in psychological well being.

If you do decide to take part you will be asked to attend GKT School of Biomedical sciences for initial assessments of:

- Cardiopulmonary fitness with measurements of oxygen uptake and heart rate.
- Muscle strength and fatigability in your lower limbs using electrical stimulation.
- Muscle spasticity in your lower limbs.
- Skin blood flow using non-invasive measurements of skin oxygenation with external loads placed on the sacrum (lower back).
- Seating pressures
- Muscle and fat thickness using ultrasound and MRI.
- Bone density using pQCT (carried out at Glasgow University).
- Mood tests using a subjective questionnaire.

You will then complete an initial period in which lower limbs are trained using FES without the tricycle. If all tests are successful you will then be provided with a tricycle to use at home. You will then follow a set protocol outlining progressive training according to your own capability.

Following this, you will carry out 52 weeks (1 year) cycling consisting of 5, 1-hour sessions each week. During this period you will be asked to keep a diary monitoring your training, health status and any problems incurred with the tricycle. You will also be required to attend KCL at 13 weeks, 26 weeks, 39 weeks and 52 weeks after the start of

training. Each visit will consist of testing as given above except for bone density measures, which will be taken only twice, once at the start (as above) then at week 52. Tests will again be taken 6 months after finishing the training programme (including bone density).

We will pay all travel expenses incurred due to participation in the study. In addition, you will be given the opportunity to keep the tricycle on completion of the study.

Only the investigators in this study will have access to the records relating to this research. The principal investigator is Professor Di Newham of Kings College London. For any further information contact Lynsey Duffell on 020 7848 6679.

We do not advise pregnant women, or those who are breast feeding to participate in the study for reasons of comfort.

In the event of your suffering any adverse effects shown to be a consequence of your participation in this study you may be compensated through the King's College London "No Fault" Compensation Scheme.

APPENDIX 2

REC Protocol No:...../.....

PARITIPANT CONSENT FORM

Title of Project

The development of Functional Electrical Stimulation (FES) systems for tricycling: improving health after spinal cord injury.

The participant should complete the whole of this sheet himself/herself.

(Please cross out as necessary)

Have you been asked to consent for yourself or on behalf of Someone else? Self/Other

If your answer to the above is "other", please give the name of the person for whom you are consenting.

Have you read the Information Sheet for Patients* and Healthy Volunteers? (This should normally be printed on the revise side of this form). Yes / No

Have you had an opportunity to ask questions and discuss this study? Yes / No

Have you received satisfactory answers to all of your questions? Yes / No

Have you received enough information about the study? Yes / No

Who have you spoken to? Prof/Dr/Mr/Ms:

Do you understand that you are free to withdraw from the study at any time, without having to give a reason for withdrawing (and without affecting your future medical care)?* Yes/ No

Do you agree to take part in this study? Yes / No

Have you declared you involvement in other research studies currently underway or undertaken in the last 12 months? Yes / No

Signed ..... Date .....

(NAME IN BLOCK LETTERS) .....

INVESTIGATOR'S STATEMENT

I confirm that I have carefully explained the nature, demands and foreseeable risks of the proposed study to the volunteer.

Signed ..... Date .....

NAME IN BLOCK LETTERS

(On completion a photocopy of this form and the information sheet should be given to the subject)

*delete or correct according to subject type

Appendix 3: Training Diary Front Cover

Cycle Training Diary

Name:

Start Date:

Instructions: Please complete this diary at every training session for the duration of the cycle training programme. This should include all information you feel is relevant to the project with reference to the guidelines outlined below.

- ¬ The trainer setting and duration of training carried out.
- ¬ Reasons for failing to complete the full protocol set for that day (if necessary).
- ¬ Any training missed, and reasons for this (eg. holiday).
- ¬ Technical or physical problems encountered.
- ¬ Suggested improvements to make training more comfortable.
- ¬ Any changes in your health-related status (including any illness, change in medication, urinary tract infections, spasticity).
- ¬ Any changes in your daily routine that may have affected your training for that day.
- ¬ Any unusual responses to the exercise.
- ¬ Any additional comments you feel would be relevant to the study.

Please use the additional sheets at the back of the diary as required (ensure you put in dates for additional comments).

Appendix 4: Training Diary Entry Sheet

Session number	Date	Time	Target for session		Duration of cycling (minutes)	Power output (watts)	Cadence (rpm)	Comments on today's session
				At target resistance				
				At reduced resistance				
				At no load				
				At target resistance				
				At reduced resistance				
				At no load				

Appendix 5: Training Diary Summary

Subject 1	week	No. Sessions	scheduled	missed	reason	compliance
	1	3	3	0		100
	2	4	3	-1		133.3333333
	3	3	3	0		100
	4	3	3	0		100
	5	3	3	0		100
	6	2	3	1		66.66666667
	7	3	3	0		100
	8	4	3	-1		133.3333333
	9	4	4	0		100
	10	5	4	-1		125
	11	3	4	1		75
	12	3	4	1		75
	13	5	4	-1		125
	14	5	4	-1		125
	15	5	4	-1		125
	16	5	4	-1		125
	17	5	5	0		100
	18	5	5	0		100
	19	0	5	5	holiday	0
	20	0	5	5	holiday	0
	21	2	5	3	equipment problems	40
	22	3	5	2	equipment problems	60
	23	5	5	0		100
	24	4	5	1		80
	25	6	5	-1		120
	26	4	5	1	unknown	80
	27	5	5	0		100
	28	5	5	0		100
	29	5	5	0		100
	30	0	5	5	holiday	0
	31	5	5	0		100
	32	5	5	0		100
	33	5	5	0		100
	34	5	5	0		100
	35	5	5	0		100
	36	5	5	0		100
	37	5	5	0		100
	38	5	5	0		100
	39	6	5	-1		120
	40	4	5	1		80
	41	6	5	-1		120
	42	3	5	2	unknown	60
	43	5	5	0		100
	44	5	5	0		100
	45	5	5	0		100
	46	4	5	1		80
	47	5	5	0		100
	48	5	5	0		100
	49	5	5	0		100
	50	4	5	1	unknown	80
	51	5	5	0		100
	52	5	5	0		100

Appendix 5: Training Diary Summary

Subject 2	week	No. sessions	scheduled	missed	reason	compliance
	1	3	3	0		100
	2	4	3	-1		133.3333333
	3	3	3	0		100
	4	3	3	0		100
	5	3	3	0		100
	6	3	3	0		100
	7	0			holiday	
	8	0			holiday	
	9	0			equipment	
	10	0			equipment	
	11	3	3	0		100
	12	4	3	-1		133.3333333
	13	4	4	0		100
	14	4	4	0		100
	15	4	4	0		100
	16	4	4	0		100
	17	0	4	4	equipment	0
	18	2	4	2	equipment	50
	19	2	4	2	equipment	50
	20	4	4	0		100
	21	4	5	1	unknown	80
	22	4	5	1	unknown	80
	23	4	5	1	unknown	80
	24	4	5	1	unknown	80
	25	4	5	1	unknown	80
	26	4	5	1	UTI	80
	27	4	5	1	unknown	80
	28	4	5	1	unknown	80
	29	4	5	1	unknown	80
	30	2	5	3	unknown	40
	31	4	5	1	unknown	80
	32	5	5	0		100
	33	5	5	0		100
	34	5	5	0		100
	35	4	5	1	unknown	80
	36	2	5	3	holiday	40
	37	3	5	2	holiday	60
	38	5	5	0		100
	39	3	5	2	skin	60
	40	6	5	-1		120
	41	5	5	0		100
	42	3	5	2	skin	60
	43				skin	
	44				skin	
	45				skin	
	46				skin	
	47				skin	
	48				skin	
	49				skin	
	50				skin	
	51				skin	
	52				restart training	
	53					
	54					
	55					
	56	2	5	3	UTI	40
	57	3	5	2	unknown	60
	58	4	5	1	unknown	80
	59	4	5	1	unknown	80
	60	4	5	1	unknown	80
	61	4	5	1	unknown	80
	62	1	5	4	equipment	20
	63	2	5	3	equipment	40
	64	5	5	0		100
	65	3	5	2	unknown	60
	66	5	5	0		100
	67	4	5	1	unknown	80
	68	5	5	0		100

Appendix 5: Training Diary Summary

Subject 3	week	No. sessions	scheduled	missed	reason	compliance
	1	3	3	0		100
	2	3	3	0		100
	3	2	3	1	equipment	66.66666667
	4	2	3	1	equipment	66.66666667
	5	2	3	1	unknown	66.66666667
	6	2	3	1	unknown	66.66666667
	7	2	3	1	unknown	66.66666667
	8	2	3	1	unknown	66.66666667
	9	2	4	2	unknown	50
	10	2	4	2	unknown	50
	11	2	4	2	unknown	50
	12	1	4	3	unknown	25
	13	3	4	1	unknown	75
	14	2	4	2	unknown	50
	15	3	4	1	unknown	75
	16	3	4	1	unknown	75
	17	3	5	2	unknown	60
	18	3	5	2	unknown	60
	19	3	5	2	unknown	60
	20	4	5	1	unknown	80
	21	4	5	1	unknown	80
	22				Family problems	
	23				Family problems	
	24				Family problems	
	25				Family problems	
	26				Family problems	
	27				Family problems	
	28				Family problems	
	29				Family problems	
	30				Family problems	
	31				Family problems	
	32				Family problems	
	33	4	5	1	unknown	80
	34	5	5	0		100
	35	4	5	1	unknown	80
	36	4	5	1	unknown	80
	37	2	5	3	unknown	40
	38	3	5	2	unknown	60
	39	5	5	0		100
	40	3	5	2	unknown	60
	41	3	5	2	unknown	60
	42	3	5	2	unknown	60
	43	2	5	3	unknown	40
	44	2	5	3	unknown	40
	45	4	5	1	unknown	80
	46	4	5	1	unknown	80
	47	2	5	3	unknown	40
	48	4	5	1	unknown	80
	49	3	5	2	unknown	60
	50	3	5	2	unknown	60
	51	4	5	1	unknown	80
	52		5			
	53	1	5	4	unknown	20
	54	3	5	2	unknown	60
	55	1	5	4	unknown	20
	56	4	5	1	unknown	80
	57	6	5	-1		120
	58	2	5	3	unknown	40
	59	0	5		holiday	
	60	0	5		holiday	
	61	0	5		holiday	
	62	0	5		holiday	
	63	3	5	2	unknown	60
	64	5	5	0		100
	65	4	5	1	unknown	80
	66	3	5	2	unknown	60
	67	3	5	2	unknown	60
	68	4	5	1	unknown	80
	69	5	5	0		100

Appendix 5: Training Diary Summary

Subject 4	week	No. sessions	scheduled	missed	reason	compliance
	1	3	3	0		100
	2	3	3	0		100
	3	4	3	-1		133.3333333
	4	2	3	1		66.66666667
	5	3	3	0		100
	6	2	3	1	unknown	66.66666667
	7	2	3	1	unknown	66.66666667
	8	2	3	1	unknown	66.66666667
	9	3	4	1	unknown	75
	10	2	4	2		50
	11	4	4	0		100
	12	3	4	1	unknown	75
	13	6	4	-2		150
	14	2	4	2	unknown	50
	15	4	4	0		100
	16	2	4	2	equipment problems	50
	17	2	5	3	equipment problems	40
	18	1	5	4	equipment problems	20
	19	5	5	0		100
	20	4	5	1	equipment problems	80
	21	4	5	1	equipment problems	80
	22	5	5	0		100
	23	3	5	2	burns	60
	24	4	5	1	burns	80
	25	4	5	1	burns	80
	26	5	5	0		100
	27	3	5	2	burns	60
	28	3	5	2	unknown	60
	29	6	5	-1		120
	30	2	5	3	UTI	40
	31	1	5	4	work	20
	32	4	5	1		80
	33	3	5	2	unknown	60
	34	3	5	2	unknown	60
	35	3	5	2	unknown	60
	36	2	5	3	equipment problems□work	40
	37	2	5	3	unknown	40
	38	4	5	1	unwell	80
	39	0	5		unwell□work	
	40	4	5	1	unknown	80
	41	2	5	3	unwell	40
	42	4	5	1	unknown	80
	43	5	5	0		100
	44	2	5	3	Holiday	40
	45	0	5		Holiday	
	46	0	5		Holiday	
	47	0	5		Holiday	
	48	0	5		Holiday	
	49	2	5	3	Holiday	40
	50	4	5	1	work	80
	51	4	5	1	injury	80
	52	3	5	2	work	60
	53	3	5	2	work	60
	54	4	5	1	unwell	80
	55	5	5	0		100
	56	5	5	0		100
	57	3	5	2	work	60
	58	2	5			

Appendix 5: Training Diary Summary

Subject 5	week	No. sessions	scheduled	missed	reason	compliance
	1	3	3	0		100
	2	3	3	0		100
	3	2	3	1	equipment	66.66666667
	4	3	3	0		100
	5	2	3	1	unknown	66.66666667
	6	2	3	1	unknown	66.66666667
	7	2	3	1	unknown	66.66666667
	8	3	3	0		100
	9	2	4	2	equipment	50
	10	2	4	2	equipment	50
	11	0				
	12	3	4	1	unknown	75
	13	4	4	0		100
	14	3	4	1	unknown	75
	15	3	4	1	unknown	75
	16	4	4	0		100
	17	5	4	-1		125
	18	4	5	1		80
	19	5	5	0		100
	20	3	5	2	unwell	60
	21	4	5	1		80
	22	6	5	-1		120
	23	6	5	-1		120
	24	5	5	0		100
	25	5	5	0		100
	26	5	5	0		100
	27	6	5	-1		120
	28	5	5	0		100
	29	6	5	-1		120
	30	3	5	2	equipment	60
	31	4	5	1		80
	32	6	5	-1		120
	33	4	5	1		80
	34	6	5	-1		120
	35	5	5	0		100
	36					
	37					
	38	4	5	1	skin	80
	39	2	5	3	unwell	40
	40	4	5	1		80
	41					
	42	4	5	1		80
	43	6	5	-1		120
	44	5	5	0		100
	45	4	5	1	unknown	80
	46	3	5	2	unknown	60
	47	4	5	1	equipment	80
	48	5	5	0		100
	49	5	5	0		100
	50	4	5	1	skin	80
	51	3	5	2	skin	60
	52	4	5	1	skin	80
	53	4	5	1	skin	80
	54	4	5	1	skin	80
	55	4	5	1	skin	80
	56	4	5	1	skin	80

Appendix 6: Matlab script for muscle CSA analysis

```
%area=GetArea(file, cal)
scnsize = get(0,'ScreenSize');
picture=imread(file,'Jpeg');
picture=squeeze(picture(:,:,1));
rowmax=max(picture');
Acal=(cal/size(picture,1))^2;
rowsum=sum(picture');

% strip out the blackish rows (2000 is the threshold) at the top and
bottom if there are any
top=min(find(rowsum>5000));
bottom=max(find(rowsum>5000));
figure(1)
pic=picture(top-2:bottom+2,1:512,1);
clf
imshow(pic)
hold on
set(gcf,'Position',round(scnsize/1))% you can change the image size
here by altering "1" to some other number
xy = []; %reset the input points
n = 0;   % and the counter
but = 1; % reset loop control
while but == 1 % Loop, picking up the points.
    [xi,yi,but] = ginput(1);
     plot(xi,yi,'.g','MarkerSize',10)
     n = n+1;
     xy(:,n) = [xi;yi];
end % of while
% Interpolate with a spline curve and finer spacing.
 if n>2
    t = 1:n;
    ts = 1: 0.1: n;
    xys = spline(t,xy,ts);
    AreacmSq=polyarea(xys(1,:),xys(2,:))*Acal
    hold off
    imshow(pic);
     hold on
     plot(xys(1,:),xys(2,:),'-y','LineWidth',1);% Plot the new curve.
     title(num2str(AreacmSq))
     hold off
 end % of if
 if n<=2  % draw a box if n=2
    cla
    [rs, cs]= size(pic);
    blowup=pic(round(rs/2):rs,round(cs/4):round(3*cs/4));
    blowup=interp2(double(blowup));
    brightest=max(max(blowup));
    blowup=blowup/brightest;
    imshow(blowup) % show enlarged part of the image
    [xx, yy]=ginput(2)


     boxwidth =(xx(2)-xx(1));
     boxheight=200;
```

```
 box=[0              boxwidth    boxwidth    0        0;....
      boxheight      boxheight   0           0        boxheight];
boxangle=atan2(yy(2)-yy(1),boxwidth);
rotbox=Rot2(box,boxangle);
hold on
 plot(rotbox(1,:)+xx(1),rotbox(2,:)+yy(1),'b','LineWidth',2)

 for k=1:2
     [x1, y1] = ginput(1);
     distance(k)=sqrt(((xx(k)-x1)^2+(yy(k)-y1)^2)*Acal)/2;
     plot([xx(k) x1],[yy(k) y1],'-r')
 end % of the k loop
 title([num2str(distance(1)) '   ' num2str(distance(2))])
     hold off

end % if
```

Appendix 7: Muscle Size - Ultrasound

	Thigh									
	Baseline		3 month		6month		9month		12month	
	Muscle	Fat	Muscle	Fat	Muscle	Fat	Muscle	Fat	Muscle	Fat
Subject										
1	1.23	1.63	1.72	1.08	2.18	1.02	2.12	1.44	2.07	1.06
2	1.32	1.18	2.45	1.1	2.84	1.13	2.83	1.18	3.1	0.91
3	1.44	1.36	2.35	1.22	2.23	0.9	2.45	0.83	2.41	1.39
4	1.09	0.36	2.07	0.42	2.12	0.45	2.58	0.3	2.52	0.37
5	1.31	0.81	2.64	0.79	2.62	0.66	3.36	0.47	3.8	0.6
average	1.278	1.068	2.246	0.922	2.398	0.832	2.668	0.844	2.78	0.866
n	0.129	0.495348	0.359	0.322056	0.31531	0.275808	0.464	0.475	0.6803308	0.397152
SD	5	5	5	5	5	5	5	5	5	5
SEM	0.058	0.221527	0.161	0.144028	0.14101	0.123345	0.207	0.213	0.30425318	0.177612

Thigh Able-Bbodied		
	Muscle	Fat
Subject		
1	2.79	0.51
2	4.14	0.46
3	2.63	0.6
4	3.62	0.88
5	2.66	1.45
average	3.168	0.78
n	0.678	0.4082
SD	5	5
SEM	0.303	0.1826

	Calf									
	0 month		3 month		6month		9 month		12 month	
	Muscle	Fat	Muscle	Fat	Muscle	Fat	Muscle	Fat	Muscle	Fat
Subject										
1	2.82	0.65	4	0.56	3.76	0.55	4.05	0.66	4.05	0.44
2	3.26	0.71	3.71	0.82	3.97	0.57	4.08	0.57	3.96	0.7
3	3.08	0.97	3.78	0.79	3.7	0.7	3.7	0.67	3.72	0.87
4	3.34	0.61	4.03	0.38	4.45	0.32	4.31	0.32	4.54	0.27
5	2.69	0.83	2.94	0.7	3.98	0.66	3.86	0.3	3.52	0.7
average	3.038	0.754	3.692	0.65	3.972	0.56	4	0.504	3.958	0.596
n	0.279	0.146561	0.442	0.181659	0.29474	0.147817	0.232	0.181	0.38590154	0.23839
SD	5	5	5	5	5	5	5	5	5	5
SEM	0.125	0.065544	0.198	0.08124	0.13181	0.066106	0.104	0.081	0.17258042	0.106611

Appendix 8: Muscle Size - MRI

Thigh & Calf

Baseline

Subject	Muscle Group	Cross-sectional area (cm^2) Right							Cross-sectional area (cm^2) Left						
		1	2	3	Average	n	sd	sem	1	2	3	Average	n	sd	sem
	Rectus femoris	2.42	2.38	2.37	2.39	3.00	0.03	0.02	1.94	2.08	2.16	2.06	3.00	0.11	0.06
	Int/lateralis	13.69	14.34	14.43	14.15	3.00	0.40	0.23	14.88	14.81	15.42	15.04	3.00	0.33	0.19
	medialis	4.30	4.27	4.25	4.27	3.00	0.03	0.01	5.70	6.12	6.00	5.94	3.00	0.22	0.12
	QUADS	**20.41**	**20.99**	**21.05**	**20.82**	**3.00**	**0.35**	**0.20**	**22.52**	**23.01**	**23.58**	**23.04**	**3.00**	**0.53**	**0.31**
	Biceps femoris long head	5.44	5.70	5.92	5.69	3.00	0.24	0.14	5.22	5.18	5.17	5.19	3.00	0.03	0.02
1	semitendinosus	3.45	3.35	3.54	3.45	3.00	0.10	0.05	3.51	3.49	3.62	3.54	3.00	0.07	0.04
	semimembranosus	4.96	5.07	4.99	5.01	3.00	0.06	0.03	4.82	5.31	5.27	5.13	3.00	0.27	0.16
	HAMS	**13.85**	**14.12**	**14.45**	**14.14**	**3.00**	**0.30**	**0.17**	**13.55**	**13.98**	**14.06**	**13.86**	**3.00**	**0.27**	**0.16**
	Gast Med	1.20	1.25	1.10	1.18	3.00	0.08	0.04	1.71	2.08	1.60	1.80	3.00	0.25	0.15
	Gast Lat														
	Soleus	10.80	10.29	11.95	11.01	3.00	0.85	0.49	13.96	14.52	13.97	14.15	3.00	0.32	0.19
	GAST/SOLEUS	**12.00**	**11.54**	**13.05**	**12.20**	**3.00**	**0.77**	**0.45**	**15.67**	**16.60**	**15.57**	**15.95**	**3.00**	**0.57**	**0.33**
	Rectus femoris	2.28	2.40	2.61	2.43	3.00	0.17	0.10	2.29	2.25	2.29	2.28	3.00	0.02	0.01
	Int/lateralis	22.13	22.38	22.52	22.34	3.00	0.20	0.11	21.27	21.20	21.05	21.17	3.00	0.11	0.06
	medialis	8.49	8.97	9.03	8.83	3.00	0.30	0.17	8.14	7.86	8.17	8.06	3.00	0.17	0.10
	QUADS	**32.90**	**33.75**	**34.16**	**33.60**	**3.00**	**0.64**	**0.37**	**31.70**	**31.31**	**31.51**	**31.51**	**3.00**	**0.20**	**0.11**
	Biceps femoris long head	7.71	7.55	7.25	7.50	3.00	0.23	0.13	6.00	5.84	6.22	6.02	3.00	0.19	0.11
2	semitendinosus	6.54	6.37	6.64	6.52	3.00	0.14	0.08	5.43	5.18	4.80	5.14	3.00	0.32	0.18
	semimembranosus	6.33	6.27	6.02	6.21	3.00	0.16	0.09	5.94	5.43	5.60	5.66	3.00	0.26	0.15
	HAMS	**20.58**	**20.19**	**19.91**	**20.23**	**3.00**	**0.34**	**0.19**	**17.37**	**16.45**	**16.62**	**16.81**	**3.00**	**0.49**	**0.28**
	Gast Med	2.14	2.44	2.30	2.29	3.00	0.15	0.09	3.27	3.21	3.28	3.25	3.00	0.04	0.02
	Gast Lat								0.47	0.43	0.63	0.51	3.00	0.11	0.06
	Soleus	15.45	15.29	14.84	15.19	3.00	0.32	0.18	15.14	14.30	14.86	14.77	3.00	0.43	0.25
	GAST/SOLEUS	**17.59**	**17.73**	**17.14**	**17.49**	**3.00**	**0.31**	**0.18**	**18.88**	**17.94**	**18.77**	**18.53**	**3.00**	**0.51**	**0.30**

	Rectus femoris	3.13	3.06	2.95	3.05	3.00	0.09	0.05	3.28	3.16	3.09	3.18	3.00	0.10	0.06
	Int/lateralis	19.03	19.85	19.00	19.29	3.00	0.48	0.28	17.16	18.00	18.30	17.82	3.00	0.59	0.34
	medialis	7.97	8.01	7.88	7.95	3.00	0.07	0.04	6.61	6.96	6.93	6.83	3.00	0.19	0.11
	QUADS	**30.13**	**30.92**	**29.83**	**30.29**	**3.00**	**0.56**	**0.33**	**27.05**	**28.12**	**28.32**	**27.83**	**3.00**	**0.68**	**0.39**
	Biceps femoris long head	3.49	3.32	3.39	3.40	3.00	0.09	0.05	6.03	6.28	5.85	6.05	3.00	0.22	0.12
3	semitendinosus	7.06	6.80	7.05	6.97	3.00	0.15	0.09	3.33	3.67	3.84	3.61	3.00	0.26	0.15
	semimembranosus	8.73	8.90	8.74	8.79	3.00	0.10	0.06	9.61	9.63	9.83	9.69	3.00	0.12	0.07
	HAMS	**19.28**	**19.02**	**19.18**	**19.16**	**3.00**	**0.13**	**0.08**	**18.97**	**19.58**	**19.52**	**19.36**	**3.00**	**0.34**	**0.19**
	Gast Med	4.19	4.20	4.01	4.13	3.00	0.11	0.06	4.81	5.17	4.84	4.94	3.00	0.20	0.12
	Gast Lat	1.03	1.07	0.97	1.02	3.00	0.05	0.03	1.76	1.60	1.61	1.66	3.00	0.09	0.05
	Soleus	23.02	22.87	22.81	22.90	3.00	0.11	0.06	19.34	19.44	19.25	19.34	3.00	0.10	0.05
	GAST/SOLEUS	**28.24**	**28.14**	**27.79**	**28.06**	**3.00**	**0.24**	**0.14**	**25.91**	**26.21**	**25.70**	**25.94**	**3.00**	**0.26**	**0.15**
	Rectus femoris	2.97	3.03	2.77	2.92	3.00	0.14	0.08	3.12	3.37	3.22	3.24	3.00	0.13	0.07
	Int/lateralis	21.21	22.45	21.90	21.85	3.00	0.62	0.36	20.11	20.80	20.98	20.63	3.00	0.46	0.27
	medialis	7.49	6.93	7.02	7.15	3.00	0.30	0.17	6.82	6.80	7.15	6.92	3.00	0.20	0.11
	QUADS	**31.67**	**32.41**	**31.69**	**31.92**	**3.00**	**0.42**	**0.24**	**30.05**	**30.97**	**31.35**	**30.79**	**3.00**	**0.67**	**0.39**
	Biceps femoris long head	6.59	6.55	6.78	6.64	3.00	0.12	0.07	6.11	6.33	6.37	6.27	3.00	0.14	0.08
4	semitendinosus	4.89	4.84	5.07	4.93	3.00	0.12	0.07	4.99	5.02	5.32	5.11	3.00	0.18	0.11
	semimembranosus	10.43	10.69	10.40	10.51	3.00	0.16	0.09	9.27	9.38	9.34	9.33	3.00	0.06	0.03
	HAMS	**21.91**	**22.08**	**22.25**	**22.08**	**3.00**	**0.17**	**0.10**	**20.37**	**20.73**	**21.03**	**20.71**	**3.00**	**0.33**	**0.19**
	Gast Med	5.34	5.16	5.40	5.30	3.00	0.12	0.07	4.57	4.86	4.81	4.75	3.00	0.16	0.09
	Gast Lat	4.67	4.98	5.12	4.92	3.00	0.23	0.13	4.23	4.36	4.91	4.50	3.00	0.36	0.21
	Soleus	16.52	17.58	16.56	16.89	3.00	0.60	0.35	15.54	14.91	15.67	15.37	3.00	0.41	0.23
	GAST/SOLEUS	**26.53**	**27.72**	**27.08**	**27.11**	**3.00**	**0.60**	**0.34**	**24.34**	**24.13**	**25.39**	**24.62**	**3.00**	**0.68**	**0.39**
5	Rectus femoris	5.06	4.92	5.10	5.03	3.00	0.09	0.05	4.72	4.61	4.84	4.72	3.00	0.12	0.07
	Int/lateralis	29.55	28.68	29.08	29.10	3.00	0.44	0.25	30.65	30.19	29.75	30.20	3.00	0.45	0.26
	medialis	8.33	8.28	8.24	8.28	3.00	0.05	0.03	10.75	11.22	11.02	11.00	3.00	0.24	0.14
	QUADS	**42.94**	**41.88**	**42.42**	**42.41**	**3.00**	**0.53**	**0.31**	**46.12**	**46.02**	**45.61**	**45.92**	**3.00**	**0.27**	**0.16**
	Biceps femoris long head	7.77	7.55	7.68	7.67	3.00	0.11	0.06	7.20	7.43	7.43	7.35	3.00	0.13	0.08
	semitendinosus	6.16	6.29	6.24	6.23	3.00	0.07	0.04	5.91	6.13	6.09	6.04	3.00	0.12	0.07
	semimembranosus	4.52	4.90	4.55	4.66	3.00	0.21	0.12	1.62	1.49	1.21	1.44	3.00	0.21	0.12

HAMS	**18.45**	**18.74**	**18.47**	**18.55**	**3.00**	**0.16**	**0.09**	**14.73**	**15.05**	**14.73**	**14.84**	**3.00**	**0.18**	**0.11**
Gast Med	5.36	5.14	5.17	5.22	3.00	0.12	0.07	5.22	5.46	5.47	5.38	3.00	0.14	0.08
Gast Lat	1.35	1.56	1.17	1.36	3.00	0.20	0.11	1.52	1.41	1.51	1.48	3.00	0.06	0.04
Soleus	14.73	14.74	15.18	14.88	3.00	0.26	0.15	14.07	14.21	13.89	14.06	3.00	0.16	0.09
GAST/SOLEUS	**21.44**	**21.44**	**21.52**	**21.47**	**3.00**	**0.05**	**0.03**	**20.81**	**21.08**	**20.87**	**20.92**	**3.00**	**0.14**	**0.08**

Subject		**Measurement (cm^2) Right**							**Measurement (cm^2) Left**						
	Thigh	**1**	**2**	**3**	**Average**	**N**	**sd**	**sem**	**1**	**2**	**3**	**Average**	**n**	**sd**	**sem**
1	muscle area	59.49	60.43	60.07	60.00	3.00	0.47	0.27	59.40	58.91	59.19	59.17	3.00	0.25	0.14
	sub. Tissue	60.51	58.88	58.61	59.33	3.00	1.03	0.59	58.83	58.35	58.68	58.62	3.00	0.25	0.14
	% muscle	49.58	50.65	50.62	50.28	3.00	0.61	0.35	50.24	50.24	50.22	50.23	3.00	0.01	0.01
	% fat	50.43	49.35	49.38	49.72	3.00	0.61	0.35	49.76	49.76	49.78	49.77	3.00	0.01	0.01
2	muscle area	77.95	77.78	78.40	78.04	3.00	0.32	0.18	84.27	83.94	84.07	84.09	3.00	0.17	0.10
	sub. Tissue	84.61	85.31	84.75	84.89	3.00	0.37	0.21	61.21	60.52	60.10	60.61	3.00	0.56	0.32
	% muscle	47.95	47.69	48.05	47.90	3.00	0.19	0.11	57.93	58.11	58.31	58.11	3.00	0.19	0.11
	% fat	52.05	52.31	51.95	52.10	3.00	0.19	0.11	42.07	41.89	41.69	41.89	3.00	0.19	0.11
3	muscle area	71.50	70.59	70.45	70.85	3.00	0.57	0.33	76.36	74.64	76.18	75.73	3.00	0.95	0.55
	sub. Tissue	55.88	56.34	55.81	56.01	3.00	0.29	0.17	42.77	44.05	42.37	43.06	3.00	0.88	0.51
	% muscle	56.13	55.61	55.80	55.85	3.00	0.26	0.15	64.10	62.89	64.26	63.75	3.00	0.75	0.43
	% fat	43.87	44.39	44.20	44.15	3.00	0.26	0.15	35.90	37.11	35.74	36.25	3.00	0.75	0.43
4	muscle area	74.91	74.62	74.16	74.56	3.00	0.38	0.22	74.48	73.38	73.47	73.78	3.00	0.61	0.35
	sub. Tissue	18.69	19.02	19.80	19.17	3.00	0.57	0.33	21.07	22.28	22.43	21.93	3.00	0.75	0.43
	% muscle	80.03	79.69	78.93	79.55	3.00	0.57	0.33	77.95	76.71	76.61	77.09	3.00	0.75	0.43
	% fat	19.97	20.31	21.07	20.45	3.00	0.57	0.33	22.05	23.29	23.39	22.91	3.00	0.75	0.43
5	muscle area	104.38	104.07	105.97	104.81	3.00	1.02	0.59	99.76	99.94	99.62	99.77	3.00	0.16	0.09

	sub. Tissue	56.94	56.71	57.06	56.90	3.00	0.18	0.10	64.87	64.95	64.07	64.63	3.00	0.49	0.28
	% muscle	64.70	64.73	65.00	64.81	3.00	0.16	0.10	60.60	60.61	60.86	60.69	3.00	0.15	0.09
	% fat	35.30	35.27	35.00	35.19	3.00	0.16	0.10	39.40	39.39	39.14	39.31	3.00	0.15	0.09
	Calf														
	muscle area	37.95	38.39	37.72	38.02	3.00	0.34	0.20	33.51	33.75	33.42	33.56	3.00	0.17	0.10
1	sub. Tissue	21.13	21.08	21.70	21.30	3.00	0.34	0.20	36.55	37.70	36.78	37.01	3.00	0.61	0.35
	% muscle	64.23	64.55	63.48	64.09	3.00	0.55	0.32	47.83	47.24	47.61	47.56	3.00	0.30	0.17
	% fat	35.77	35.45	36.52	35.91	3.00	0.55	0.32	52.17	52.76	52.39	52.44	3.00	0.30	0.17
	muscle area	50.31	49.79	49.86	49.99	3.00	0.28	0.16	47.66	48.67	48.62	48.32	3.00	0.57	0.33
2	sub. Tissue	28.67	29.31	29.66	29.21	3.00	0.50	0.29	32.74	32.12	32.05	32.30	3.00	0.38	0.22
	% muscle	63.70	62.95	62.70	63.12	3.00	0.52	0.30	59.28	60.24	60.27	59.93	3.00	0.56	0.33
	% fat	36.30	37.05	37.30	36.88	3.00	0.52	0.30	40.72	39.76	39.73	40.07	3.00	0.56	0.33
	muscle area	53.97	54.28	54.62	54.29	3.00	0.33	0.19	57.08	56.95	56.10	56.71	3.00	0.53	0.31
3	sub. Tissue	24.62	24.20	24.04	24.29	3.00	0.30	0.17	20.29	19.76	20.99	20.35	3.00	0.62	0.36
	% muscle	68.67	69.16	69.44	69.09	3.00	0.39	0.22	73.78	74.24	72.77	73.60	3.00	0.75	0.43
	% fat	31.33	30.84	30.56	30.91	3.00	0.39	0.22	26.22	25.76	27.23	26.40	3.00	0.75	0.43
	muscle area	54.13	54.77	54.33	54.41	3.00	0.33	0.19	55.20	54.97	55.33	55.17	3.00	0.18	0.11
4	sub. Tissue	15.03	14.94	15.55	15.17	3.00	0.33	0.19	13.12	13.49	13.38	13.33	3.00	0.19	0.11
	% muscle	78.27	78.57	77.75	78.19	3.00	0.42	0.24	80.80	80.30	80.53	80.54	3.00	0.25	0.14
	% fat	21.73	21.43	22.25	21.81	3.00	0.42	0.24	19.20	19.70	19.47	19.46	3.00	0.25	0.14
	muscle area	49.96	49.43	49.11	49.50	3.00	0.43	0.25	51.48	51.01	51.40	51.30	3.00	0.25	0.15
5	sub. Tissue	29.35	30.09	30.83	30.09	3.00	0.74	0.43	25.17	25.41	25.28	25.29	3.00	0.12	0.07
	% muscle	62.99	62.16	61.43	62.20	3.00	0.78	0.45	67.16	66.75	67.03	66.98	3.00	0.21	0.12
	% fat	37.01	37.84	38.57	37.80	3.00	0.78	0.45	32.84	33.25	32.97	33.02	3.00	0.21	0.12

12 Month

Subject	Muscle Group	Cross-sectional area (cm^2) Right							Cross-sectional area (cm^2) Left						
		1	2	3	Average	n	sd	sem	1	2	3	Average	n	sd	sem
	Rectus femoris	3.67	3.50	3.38	3.52	3.00	0.15	0.08	4.78	4.74	4.74	4.75	3.00	0.02	0.01
	Int/lateralis	26.95	26.86	26.51	26.77	3.00	0.23	0.13	28.69	29.15	28.48	28.77	3.00	0.34	0.20
	medialis	7.28	6.52	6.75	6.85	3.00	0.39	0.23	5.65	5.45	5.45	5.52	3.00	0.12	0.07
	QUADS	**37.90**	**36.88**	**36.64**	**37.14**	**3.00**	**0.67**	**0.39**	**39.12**	**39.34**	**38.67**	**39.04**	**3.00**	**0.34**	**0.20**
	Biceps femoris long head	5.37	4.99	5.42	5.26	3.00	0.24	0.14	4.84	5.15	5.18	5.06	3.00	0.19	0.11
1	semitendinosus	5.62	5.84	5.94	5.80	3.00	0.16	0.09	6.31	6.31	6.23	6.28	3.00	0.05	0.03
	semimembranosus	5.96	5.87	5.96	5.93	3.00	0.05	0.03	5.41	5.42	5.30	5.38	3.00	0.07	0.04
	HAMS	**16.95**	**16.70**	**17.32**	**16.99**	**3.00**	**0.31**	**0.18**	**16.56**	**16.88**	**16.71**	**16.72**	**3.00**	**0.16**	**0.09**
	Gast Med	1.97	2.45	2.38	2.27	3.00	0.26	0.15	3.67	3.76	3.56	3.66	3.00	0.10	0.06
	Gast Lat														
	Soleus	17.85	17.79	17.32	17.65	3.00	0.29	0.17	16.15	16.19	16.10	16.15	3.00	0.05	0.03
	GAST/SOLEUS	**19.82**	**20.24**	**19.70**	**19.92**	**3.00**	**0.28**	**0.16**	**19.82**	**19.95**	**19.66**	**19.81**	**3.00**	**0.15**	**0.08**
	Rectus femoris	4.57	4.09	4.40	4.35	3.00	0.24	0.14	3.79	3.65	3.83	3.76	3.00	0.09	0.05
	Int/lateralis	35.96	35.99	35.78	35.91	3.00	0.11	0.07	31.26	31.37	32.09	31.57	3.00	0.45	0.26
	medialis	9.59	10.50	9.37	9.82	3.00	0.60	0.35	12.90	12.77	13.14	12.94	3.00	0.19	0.11
	QUADS	**50.12**	**50.58**	**49.55**	**50.08**	**3.00**	**0.52**	**0.30**	**47.95**	**47.79**	**49.06**	**48.27**	**3.00**	**0.69**	**0.40**
	Biceps femoris long head	8.14	7.82	7.77	7.91	3.00	0.20	0.12	9.73	9.04	9.50	9.42	3.00	0.35	0.20
2	semitendinosus	9.36	9.71	10.12	9.73	3.00	0.38	0.22	7.82	7.28	7.61	7.57	3.00	0.27	0.16
	semimembranosus	7.15	7.32	7.16	7.21	3.00	0.10	0.06	6.81	7.04	7.04	6.96	3.00	0.13	0.08
	HAMS	**24.65**	**24.85**	**25.05**	**24.85**	**3.00**	**0.20**	**0.12**	**24.36**	**23.36**	**24.15**	**23.96**	**3.00**	**0.53**	**0.30**
	Gast Med	2.72	2.43	2.71	2.62	3.00	0.16	0.10	4.16	4.05	3.78	4.00	3.00	0.20	0.11
	Gast Lat								2.03	2.13	1.96	2.04	3.00	0.09	0.05
	Soleus	21.81	20.72	20.52	21.02	3.00	0.69	0.40	21.06	20.27	21.38	20.90	3.00	0.57	0.33
	GAST/SOLEUS	**24.53**	**23.15**	**23.23**	**23.64**	**3.00**	**0.77**	**0.45**	**27.25**	**26.45**	**27.12**	**26.94**	**3.00**	**0.43**	**0.25**

	Rectus femoris	4.43	4.01	4.54	4.33	3.00	0.28	0.16	3.71	3.83	4.22	3.92	3.00	0.27	0.15
	Int/lateralis	22.61	22.96	21.80	22.46	3.00	0.60	0.34	19.92	20.73	20.48	20.38	3.00	0.41	0.24
	medialis	10.84	10.60	11.13	10.86	3.00	0.27	0.15	8.51	8.89	8.78	8.73	3.00	0.20	0.11
	QUADS	**37.88**	**37.57**	**37.47**	**37.64**	**3.00**	**0.21**	**0.12**	**32.14**	**33.45**	**33.48**	**33.02**	**3.00**	**0.77**	**0.44**
	Biceps femoris long head	7.20	7.34	7.01	7.18	3.00	0.17	0.10	8.12	8.39	8.15	8.22	3.00	0.15	0.09
3	semitendinosus	4.98	5.21	5.07	5.09	3.00	0.12	0.07	3.34	3.49	3.47	3.43	3.00	0.08	0.05
	semimembranosus	13.30	13.82	14.10	13.74	3.00	0.41	0.23	13.00	12.85	13.74	13.20	3.00	0.48	0.28
	HAMS	**25.48**	**26.37**	**26.18**	**26.01**	**3.00**	**0.47**	**0.27**	**24.46**	**24.73**	**25.36**	**24.85**	**3.00**	**0.46**	**0.27**
	Gast Med	4.35	4.57	4.33	4.42	3.00	0.13	0.08	3.52	3.77	3.20	3.50	3.00	0.29	0.16
	Gast Lat	1.81	1.57	1.84	1.74	3.00	0.15	0.09	1.07	0.87	0.84	0.93	3.00	0.13	0.07
	Soleus	18.12	18.65	17.79	18.19	3.00	0.43	0.25	18.15	19.10	19.31	18.85	3.00	0.62	0.36
	GAST/SOLEUS	**24.28**	**24.79**	**23.96**	**24.34**	**3.00**	**0.42**	**0.24**	**22.74**	**23.74**	**23.35**	**23.28**	**3.00**	**0.50**	**0.29**
	Rectus femoris	6.91	7.42	7.28	7.20	3.00	0.26	0.15	5.85	5.95	6.25	6.02	3.00	0.21	0.12
	Int/lateralis	36.37	37.42	37.10	36.96	3.00	0.54	0.31	39.73	39.01	38.67	39.14	3.00	0.54	0.31
	medialis	11.10	11.55	12.21	11.62	3.00	0.56	0.32	9.84	10.10	10.42	10.12	3.00	0.29	0.17
	QUADS	**54.38**	**56.39**	**56.59**	**55.79**	**3.00**	**1.22**	**0.71**	**55.42**	**55.06**	**55.34**	**55.27**	**3.00**	**0.19**	**0.11**
	Biceps femoris long head	8.01	7.82	7.82	7.88	3.00	0.11	0.06	8.49	8.43	8.64	8.52	3.00	0.11	0.06
4	semitendinosus	7.87	8.11	8.48	8.15	3.00	0.31	0.18	6.21	6.53	6.12	6.29	3.00	0.22	0.12
	semimembranosus	9.95	10.40	9.86	10.07	3.00	0.29	0.17	13.12	13.35	13.57	13.35	3.00	0.23	0.13
	HAMS	**25.83**	**26.33**	**26.16**	**26.11**	**3.00**	**0.25**	**0.15**	**27.82**	**28.31**	**28.33**	**28.15**	**3.00**	**0.29**	**0.17**
	Gast Med	7.62	7.35	8.11	7.69	3.00	0.39	0.22	7.94	7.33	7.69	7.65	3.00	0.31	0.18
	Gast Lat	4.21	3.99	4.27	4.16	3.00	0.15	0.09	7.25	7.04	7.14	7.14	3.00	0.11	0.06
	Soleus	23.15	24.95	24.13	24.08	3.00	0.90	0.52	25.45	25.18	23.80	24.81	3.00	0.89	0.51
	GAST/SOLEUS	**34.98**	**36.29**	**36.51**	**35.93**	**3.00**	**0.83**	**0.48**	**40.64**	**39.55**	**38.63**	**39.61**	**3.00**	**1.01**	**0.58**
5	Rectus femoris	3.35	3.26	3.17	3.26	3.00	0.09	0.05	5.92	6.25	6.12	6.10	3.00	0.17	0.10
	Int/lateralis	28.56	29.02	29.38	28.99	3.00	0.41	0.24	39.77	39.10	39.06	39.31	3.00	0.40	0.23
	medialis	22.99	22.85	23.08	22.97	3.00	0.12	0.07	21.62	21.36	21.02	21.33	3.00	0.30	0.17
	QUADS	**54.90**	**55.13**	**55.63**	**55.22**	**3.00**	**0.37**	**0.22**	**67.31**	**66.71**	**66.20**	**66.74**	**3.00**	**0.56**	**0.32**
	Biceps femoris long head	8.89	8.68	8.14	8.57	3.00	0.39	0.22	8.57	8.81	8.74	8.71	3.00	0.12	0.07
	semitendinosus	7.34	6.78	7.47	7.20	3.00	0.37	0.21	5.26	5.27	5.00	5.18	3.00	0.15	0.09
	semimembranosus	12.69	12.02	12.34	12.35	3.00	0.34	0.19	5.72	5.48	5.48	5.56	3.00	0.14	0.08

HAMS	**28.92**	**27.48**	**27.95**	**28.12**	**3.00**	**0.73**	**0.42**	**19.55**	**19.56**	**19.22**	**19.44**	**3.00**	**0.19**	**0.11**
Gast Med	4.62	5.04	4.35	4.67	3	0.348	0.201	5.55	5.56	5.72	5.61	3	0.0954	0.055
Gast Lat	0.82	0.71	0.88	0.80333	3	0.086	0.05	1.46	2.19	1.24	1.63	3	0.4973	0.287
Soleus	24.72	24.48	24.41	24.5367	3	0.163	0.094	22.43	21.86	21.97	22.0867	3	0.3024	0.175
GAST/SOLEUS	**30.16**	**30.23**	**29.64**	**30.01**	**3**	**0.322**	**0.186**	**29.44**	**29.61**	**28.93**	**29.3267**	**3**	**0.3539**	**0.204**

Subject		**Measurement (cm^2) Right**							**Measurement (cm^2) Left**						
	Thigh	**1**	**2**	**3**	**Average**	**n**	**sd**	**sem**	**1**	**2**	**3**	**Average**	**n**	**sd**	**sem**
1	muscle area	88.00	87.62	88.28	87.97	3.00	0.33	0.19	96.37	96.30	96.75	96.47	3.00	0.24	0.14
	sub. Tissue	56.45	56.66	56.92	56.68	3.00	0.24	0.14	66.48	67.05	65.32	66.28	3.00	0.88	0.51
	% muscle	60.92	60.73	60.80	60.82	3.00	0.10	0.06	59.18	58.95	59.70	59.28	3.00	0.38	0.22
	% fat	39.08	39.27	39.20	39.18	3.00	0.10	0.06	40.82	41.05	40.30	40.72	3.00	0.38	0.22
2	muscle area	113.28	113.14	113.48	113.30	3.00	0.17	0.10	112.14	112.44	112.32	112.30	3.00	0.15	0.09
	sub. Tissue	69.48	69.40	69.37	69.42	3.00	0.06	0.03	77.06	73.45	73.77	74.76	3.00	2.00	1.15
	% muscle	61.98	61.98	62.06	62.01	3.00	0.05	0.03	59.27	60.49	60.36	60.04	3.00	0.67	0.39
	% fat	38.02	38.02	37.94	37.99	3.00	0.05	0.03	40.73	39.51	39.64	39.96	3.00	0.67	0.39
3	muscle area	87.51	87.16	86.71	87.13	3.00	0.40	0.23	88.24	88.96	88.23	88.48	3.00	0.42	0.24
	sub. Tissue	45.75	45.70	45.85	45.77	3.00	0.08	0.04	45.42	44.30	45.49	45.07	3.00	0.67	0.39
	% muscle	65.67	65.60	65.41	65.56	3.00	0.13	0.08	66.02	66.76	65.98	66.25	3.00	0.44	0.25
	% fat	34.33	34.40	34.59	34.44	3.00	0.13	0.08	33.98	33.24	34.02	33.75	3.00	0.44	0.25
4	muscle area	110.96	110.79	111.15	110.97	3.00	0.18	0.10	113.75	113.68	113.18	113.54	3.00	0.31	0.18
	sub. Tissue	22.04	21.41	21.06	21.50	3.00	0.50	0.29	18.84	18.94	19.98	19.25	3.00	0.63	0.36
	% muscle	83.43	83.80	84.07	83.77	3.00	0.32	0.19	85.79	85.72	85.00	85.50	3.00	0.44	0.25
	% fat	16.57	16.20	15.93	16.23	3.00	0.32	0.19	14.21	14.28	15.00	14.50	3.00	0.44	0.25
5	muscle area	122.60	123.26	122.59	122.82	3.00	0.38	0.22	133.98	133.30	133.62	133.63	3.00	0.34	0.20

	sub. Tissue	38.19	36.26	37.19	37.21	3.00	0.97	0.56	38.50	39.26	38.71	38.82	3.00	0.39	0.23
	% muscle	76.25	77.27	76.72	76.75	3.00	0.51	0.29	77.68	77.25	77.54	77.49	3.00	0.22	0.13
	% fat	23.75	22.73	23.28	23.25	3.00	0.51	0.29	22.32	22.75	22.46	22.51	3.00	0.22	0.13
	Calf														
	muscle area	43.45	44.12	44.27	43.95	3.00	0.44	0.25	46.63	46.26	47.24	46.71	3.00	0.49	0.29
1	sub. Tissue	22.06	21.77	21.63	21.82	3.00	0.22	0.13	24.69	24.76	23.43	24.29	3.00	0.75	0.43
	% muscle	66.33	66.96	67.18	66.82	3.00	0.44	0.26	65.38	65.14	66.85	65.79	3.00	0.92	0.53
	% fat	33.67	33.04	32.82	33.18	3.00	0.44	0.26	34.62	34.86	33.15	34.21	3.00	0.92	0.53
	muscle area	53.69	54.23	54.45	54.12	3.00	0.39	0.23	60.07	60.01	60.89	60.32	3.00	0.49	0.28
2	sub. Tissue	32.21	30.35	30.24	30.93	3.00	1.11	0.64	28.93	27.07	26.38	27.46	3.00	1.32	0.76
	% muscle	62.50	64.12	64.29	63.64	3.00	0.99	0.57	67.49	68.91	69.77	68.73	3.00	1.15	0.66
	% fat	37.50	35.88	35.71	36.36	3.00	0.99	0.57	32.51	31.09	30.23	31.27	3.00	1.15	0.66
	muscle area	53.08	53.43	52.67	53.06	3.00	0.38	0.22	52.59	52.98	52.66	52.74	3.00	0.21	0.12
3	sub. Tissue	17.22	18.45	18.21	17.96	3.00	0.65	0.38	19.67	18.45	19.16	19.09	3.00	0.61	0.35
	% muscle	75.50	74.33	74.31	74.72	3.00	0.68	0.39	72.78	74.17	73.32	73.42	3.00	0.70	0.40
	% fat	24.50	25.67	25.69	25.28	3.00	0.68	0.39	27.22	25.83	26.68	26.58	3.00	0.70	0.40
	muscle area	70.21	70.40	70.53	70.38	3.00	0.16	0.09	71.33	72.00	71.14	71.49	3.00	0.45	0.26
4	sub. Tissue	13.71	13.68	14.32	13.90	3.00	0.36	0.21	15.41	14.44	15.39	15.08	3.00	0.55	0.32
	% muscle	83.66	83.73	83.12	83.51	3.00	0.33	0.19	82.23	83.29	82.21	82.58	3.00	0.62	0.36
	% fat	16.34	16.27	16.88	16.49	3.00	0.33	0.19	17.77	16.71	17.79	17.42	3.00	0.62	0.36
	muscle area	58.52	58.17	58.98	58.56	3.00	0.41	0.23	58.47	57.99	57.67	58.04	3.00	0.40	0.23
5	sub. Tissue	24.17	25.31	23.78	24.42	3.00	0.80	0.46	22.60	23.22	24.10	23.31	3.00	0.75	0.44
	% muscle	70.77	69.68	71.27	70.57	3.00	0.81	0.47	72.12	71.41	70.53	71.35	3.00	0.80	0.46
	% fat	29.23	30.32	28.73	29.43	3.00	0.81	0.47	27.88	28.59	29.47	28.65	3.00	0.80	0.46

Appendix 8: Muscle Size: MRI

Gluteals

Baseline Subject		Right							Left						
		1	2	3	Average	n	sd	sem	1	2	3	Average	n	sd	sem
	Ischeum-fat	2.21	2.18	2.21	2.2	3	0.0173	0.01	2.18	2.21	2.27	2.22	3	0.046	0.0265
1	Ischeum-muscle	0.6	0.76	0.63	0.663333	3	0.085	0.05	0.6	0.82	0.63	0.68333	3	0.119	0.0689
	muscle-fat	1.61	1.42	1.58	1.536667	3	0.1021	0.06	1.58	1.39	1.64	1.53667	3	0.131	0.0754
	Ischeum-fat	3.7	3.65	3.73	3.693333	3	0.0404	0.02	3.51	3.44	3.51	3.48667	3	0.04	0.0233
2	Ischeum-muscle	1.8	1.85	1.83	1.826667	3	0.0252	0.01	1.52	1.52	1.54	1.52667	3	0.012	0.0067
	muscle-fat	1.9	1.8	1.9	1.866667	3	0.0577	0.03	1.99	1.92	1.97	1.96	3	0.036	0.0208
	Ischeum-fat	3.11	3.07	3.03	3.07	3	0.04	0.02	3.36	3.36	3.32	3.34667	3	0.023	0.0133
3	Ischeum-muscle	1.52	1.44	1.35	1.436667	3	0.085	0.05	1.72	1.76	1.68	1.72	3	0.04	0.0231
	muscle-fat	1.59	1.63	1.68	1.633333	3	0.0451	0.03	1.64	1.6	1.64	1.62667	3	0.023	0.0133
	Ischeum-fat	1.01	0.98	0.95	0.98	3	0.03	0.02	0.73	0.69	0.66	0.69333	3	0.035	0.0203
4	Ischeum-muscle	0.32	0.35	0.41	0.36	3	0.0458	0.03	0.38	0.35	0.47	0.4	3	0.062	0.0361
	muscle-fat	0.69	0.63	0.54	0.62	3	0.0755	0.04	0.35	0.34	0.19	0.29333	3	0.09	0.0517
	Ischeum-fat	3.53	3.5	3.5	3.51	3	0.0173	0.01	3.47	3.37	3.47	3.43667	3	0.058	0.0333
5	Ischeum-muscle	1.51	1.55	1.48	1.513333	3	0.0351	0.02	1.77	1.83	1.8	1.8	3	0.03	0.0173
	muscle-fat	2.02	1.95	2.02	1.996667	3	0.0404	0.02	1.7	1.54	1.67	1.63667	3	0.085	0.0491
1	Gluteal area	15.2	14.7	14	14.65667	3	0.6062	0.35	15.4	15.73	16.4	15.8567	3	0.492	0.2843
2	Gluteal area	33.5	31.35	32.8	32.57	3	1.1099	0.64	26.5	25.63	27.18	26.4433	3	0.778	0.4491
3	Gluteal area	30.2	30.04	29.2	29.78667	3	0.5369	0.31	31.7	30.93	31.55	31.3767	3	0.39	0.2252
4	Gluteal area	17.8	18.11	19.3	18.40333	3	0.7824	0.45	25.8	25.14	24.75	25.24	3	0.547	0.3158
5	Gluteal area	24.5	25.73	25.5	25.22667	3	0.6747	0.39	29.3	29.92	29.52	29.5833	3	0.31	0.1789

Appendix 8: Muscle Size: MRI

Gluteals

12 Month Subject		Right							Left						
		1	2	3	Average	n	sd	sem	1	2	3	Average	n	sd	sem
	Ischeum-fat	2.33	2.37	2.37	2.356667	3	0.0231	0.01	2.96	2.86	2.99	2.93667	3	0.068	0.0393
1	Ischeum-muscle	1	0.97	1	0.99	3	0.0173	0.01	1.28	1.25	1.21	1.24667	3	0.035	0.0203
	muscle-fat	1.33	1.4	1.37	1.366667	3	0.0351	0.02	1.68	1.61	1.78	1.69	3	0.085	0.0493
	Ischeum-fat	3.44	3.44	3.4	3.426667	3	0.0231	0.01	3.94	3.9	3.83	3.89	3	0.056	0.0321
2	Ischeum-muscle	1.52	1.45	1.48	1.483333	3	0.0351	0.02	1.48	1.59	1.55	1.54	3	0.056	0.0321
	muscle-fat	1.92	1.99	1.92	1.943333	3	0.0404	0.02	2.46	2.31	2.28	2.35	3	0.096	0.0557
	Ischeum-fat	2.14	2.11	2.11	2.12	3	0.0173	0.01	2.38	2.48	2.45	2.43667	3	0.051	0.0296
3	Ischeum-muscle	0.97	0.93	0.97	0.956667	3	0.0231	0.01	1.1	1.07	1.07	1.08	3	0.017	0.01
	muscle-fat	1.17	1.18	1.14	1.163333	3	0.0208	0.01	1.28	1.41	1.38	1.35667	3	0.068	0.0393
	Ischeum-fat	0.62	0.56	0.59	0.59	3	0.03	0.02	0.84	0.84	0.87	0.85	3	0.017	0.01
4	Ischeum-muscle	0.31	0.44	0.4	0.383333	3	0.0666	0.04	0.47	0.44	0.41	0.44	3	0.03	0.0173
	muscle-fat	0.31	0.12	0.19	0.206667	3	0.0961	0.06	0.37	0.4	0.46	0.41	3	0.046	0.0265
	Ischeum-fat	2.85	2.79	2.88	2.84	3	0.0458	0.03	3.02	2.98	3.05	3.01667	3	0.035	0.0203
5	Ischeum-muscle	2.2	2.1	2.1	2.133333	3	0.0577	0.03	1.7	1.67	1.7	1.69	3	0.017	0.01
	muscle-fat	0.65	0.69	0.78	0.706667	3	0.0666	0.04	1.32	1.31	1.35	1.32667	3	0.021	0.012
1	Gluteal area	24.5	24.24	24	24.24	3	0.29	0.17	29.6	29.82	29.8	29.7233	3	0.15	0.0869
2	Gluteal area	37.4	37.45	36.6	37.17	3	0.4763	0.28	39.2	38.65	38.28	38.72	3	0.479	0.2765
3	Gluteal area	28.6	28.36	26.4	27.78333	3	1.1944	0.69	32	31.61	31.89	31.8267	3	0.193	0.1114
4	Gluteal area	19.6	21.13	20.5	20.42333	3	0.7596	0.44	26	26.02	25.64	25.89	3	0.217	0.125
5	Gluteal area	33	32.69	32.7	32.78333	3	0.1447	0.08	33.7	33.97	33.33	33.68	3	0.324	0.1872

Appendix 9: Contractile Properties

SCI People

	Lever	Max Toque v	Max Toque N	Max Toque Nm	Torque:Frequency (Nm) 1 Hz	10 Hz	20 Hz	50 Hz	100 Hz	Twitch Time (ms) 0.5 Contr	0.5 Relax	Fatigue Resistance (Nm) Initial	1 Min	2 Min	3 Min
BASELINE															
Subject															
1	0.31	0.13	50.7	15.72	4.23	4.96	5.8	7.37	7.01	50	30	6.41	2.66	1.45	1.09
2	0.28	0.19	74.1	20.75	1.53	2.4	9.5	10.7	11.36	46	61	11.14	6.22	2.84	2.29
3	0.3	0.09	35.1	10.53	1.87	2.34	2.57	2.46	2.46	28	39	4.33	1.52	1.52	1.52
4	0.37	0.12	46.8	17.32	1.59	1.73	4.33	6.93	7.07	36	85	5.92	3.9	2.6	2.02
5	0.31	0.04	15.6	4.84	0.97	1.09	1.21	1.93	1.93	32	46	1.09	0.73	0.24	0.24
average	0.31	0.11	44.46	13.83	2.04	2.5	4.68	5.88	5.97	38.4	52.2	5.78	3.01	1.73	1.43
n	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5
SD	0.03	0.06	21.47	6.23	1.27	1.47	3.21	3.67	3.87	9.32	21.56	3.65	2.16	1.04	0.81
SEM	0.02	0.02	9.6	2.79	0.57	0.66	1.43	1.64	1.73	4.17	9.64	1.63	0.97	0.46	0.36
3 MONTH															
Subject															
1	0.31	0.32	124.8	38.69	3.63	4.96	6.17	7.98	9.55	51	30	8.58	5.68	4.47	4.11
2	0.3	0.55	214.5	64.35	3.28	5.97	11.35	15.91	18.72	81	51	14.98	14.16	13.22	12.05
3	0.32	0.39	152.1	48.67	4.74	4.87	11.11	14.98	16.1	45	71	19.84	14.35	11.86	11.11
4	0.37	0.46	179.4	66.38	9.24	17.32	25.97	31.75	33.19	30	54	30.3	21.65	15.87	14.43
5	0.32	1.02	397.8	127.3	8.86	10.11	22.84	30.83	32.57	50	42	25.08	19.84	13.73	11.73
average	0.32	0.55	213.7	69.08	5.95	8.64	15.49	20.29	22.03	51.4	49.6	19.76	15.14	11.83	10.69
n	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5
SD	0.03	0.28	108.1	34.49	2.88	5.3	8.47	10.5	10.46	18.56	15.18	8.48	6.23	4.36	3.89
SEM	0.01	0.12	48.36	15.43	1.29	2.37	3.79	4.7	4.68	8.3	6.79	3.79	2.79	1.95	1.74

Appendix 9: Contractile Properties

SCI People

6 MONTH															
Subject															
1	**0.33**	**0.34**	**132.6**	**43.76**	**4.76**	**5.02**	**9.52**	**10.81**	**14.41**	**66**	**20**	**14.29**	**10.17**	**7.59**	**6.31**
2	**0.32**	**0.53**	**206.7**	**66.14**	**5.74**	**9.98**	**15.35**	**19.22**	**22.34**	**41**	**65**	**17.22**	**14.73**	**13.23**	**12.6**
3	**0.31**	**0.48**	**187.2**	**58.03**	**7.25**	**3.39**	**11.24**	**15.84**	**17.65**	**23**	**41**	**15.35**	**15.11**	**11.85**	**11.61**
4															
5	**0.3**	**1.27**	**495.3**	**148.6**	**12.6**	**25.96**	**40.93**	**50.42**	**52.04**	**60**	**64**	**44.05**	**31.7**	**25.96**	**24.09**
average	**0.32**	**0.66**	**255.5**	**79.13**	**7.59**	**11.09**	**19.26**	**24.07**	**26.61**	**47.5**	**47.5**	**22.73**	**17.93**	**14.66**	**13.65**
n	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**	**4**
SD	**0.01**	**0.42**	**163**	**47.22**	**3.5**	**10.3**	**14.65**	**17.9**	**17.26**	**19.5**	**21.42**	**14.27**	**9.45**	**7.91**	**7.49**
SEM	**0.01**	**0.21**	**81.47**	**23.61**	**1.75**	**5.15**	**7.33**	**8.95**	**8.63**	**9.75**	**10.71**	**7.13**	**4.73**	**3.95**	**3.74**
9 MONTH															
Subject															
1	**0.3**	**0.35**	**136.5**	**40.95**	**2.46**	**4.45**	**7.02**	**10.53**	**13.81**	**51**	**35**	**8.54**	**8.42**	**6.79**	**6.08**
2	**0.34**	**0.65**	**253.5**	**86.19**	**5.7**	**9.41**	**22.01**	**25.99**	**30.5**	**54**	**25**	**21.35**	**19.62**	**18.83**	**18.83**
3	**0.32**	**0.44**	**171.6**	**54.91**	**9.86**	**10.23**	**15.48**	**19.59**	**22.34**	**23**	**49**	**21.59**	**18.35**	**14.35**	**13.85**
4	**0.39**	**0.49**	**191.1**	**74.53**	**5.32**	**7.15**	**10.34**	**16.12**	**17.64**	**24**	**33**	**22.05**	**18.1**	**12.32**	**12.02**
5	**0.32**	**1.3**	**507**	**162.2**	**17.47**	**31.2**	**48.67**	**57.41**	**62.4**	**46**	**53**	**56.16**	**42.43**	**36.19**	**33.7**
average	**0.33**	**0.65**	**251.9**	**83.76**	**8.16**	**12.49**	**20.7**	**25.93**	**29.34**	**39.6**	**39**	**25.94**	**21.39**	**17.7**	**16.9**
n	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**
SD	**0.03**	**0.38**	**148.8**	**47.21**	**5.84**	**10.7**	**16.63**	**18.47**	**19.5**	**14.98**	**11.66**	**17.83**	**12.59**	**11.21**	**10.44**
SEM	**0.02**	**0.17**	**66.54**	**21.11**	**2.61**	**4.78**	**7.44**	**8.26**	**8.72**	**6.7**	**5.22**	**7.97**	**5.63**	**5.01**	**4.67**
12 MONTH															
Subject															
1	**0.31**	**0.39**	**152.1**	**47.15**	**3.14**	**7.25**	**9.43**	**14.15**	**19.83**	**46**	**39**	**9.19**	**7.98**	**7.62**	**7.37**
2	**0.34**	**0.99**	**386.1**	**131.3**	**8.35**	**14.19**	**22.54**	**27.05**	**29.44**	**47**	**65**	**24.13**	**22.01**	**20.95**	**19.62**
3	**0.3**	**0.78**	**304.2**	**91.26**	**8.89**	**5.73**	**16.38**	**35.1**	**40.13**	**28**	**40**	**33.58**	**29.13**	**20.59**	**17.43**
4	**0.37**	**0.79**	**308.1**	**114**	**7.5**	**10.25**	**24.53**	**41.85**	**43.87**	**27**	**68**	**30.3**	**24.96**	**22.94**	**21.36**
5	**0.34**	**1.14**	**444.6**	**151.2**	**11.14**	**14.19**	**27.71**	**35.93**	**42.03**	**66**	**41**	**35.4**	**30.1**	**25.59**	**25.86**
average	**0.33**	**0.82**	**319**	**107**	**7.81**	**10.32**	**20.12**	**30.82**	**35.06**	**42.8**	**50.6**	**26.52**	**22.84**	**19.54**	**18.33**
n	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**
SD	**0.03**	**0.28**	**110.1**	**40.06**	**2.93**	**3.88**	**7.27**	**10.7**	**10.19**	**16.08**	**14.57**	**10.6**	**8.92**	**6.95**	**6.86**
SEM	**0.01**	**0.13**	**49.23**	**17.91**	**1.31**	**1.74**	**3.25**	**4.79**	**4.56**	**7.19**	**6.52**	**4.74**	**3.99**	**3.11**	**3.07**

Able-Bodied

		Max Toque			Torque:Frequency (Nm)					Twitch Time (ms)		Fatigue Resistance (Nm)			
	Lever	v	N	Nm	1 Hz	10 Hz	20 Hz	50 Hz	100 Hz	0.5 Contr	0.5 Relax	Initial	1 Min	2 Min	3 Min
Subject															
1	0.37	1.96	765.6	283.3	16.88	19.05	43.72	52.09	58.44	6	26	79.22	65.8	52.09	48.2
2	0.37	1.34	523.8	193.8	16.59	26.41	36.65	44.44	45.74	37	42	41.27	36.22	33.77	33.48
3	0.38	1.99	776.1	294.9	10.52	29.34	41.64	54.69	55.72	29	45	42.68	27.86	24.6	22.08
4	0.39	1.96	763.6	297.8	8.82	22.21	35.59	42.59	41.98	29	46	38.94	34.53	29.36	27.83
5	0.39	2.37	925.5	360.9	16.73	35.29	51.56	63.12	67.53	44	79	52.02	38.63	37.87	33.01
6	0.35	3.88	1511	528.9	15.7	19.66	76.71	113.98	136	45	64	86.27	76.03	59.92	59.38
7	0.34	2.21	862.7	293.3	6.23	17.77	30.5	47.07	51.05	40	47	28.91	22.01	16.71	15.78
8	0.32	3.07	1197	383.1	11.23	26.58	51.67	70.26	77	45	44	75.63	62.15	54.66	55.16
9	0.28	3.67	1431	400.7	12.23	32.21	63.34	92.6	109.2	50	52	64.76	53.51	38.66	38.88
10	0.28	3.42	1334	373.6	9.61	35.71	54.82	68.47	74.8	25	69	52.96	36.69	30.03	29.92
average	0.33	2.59	1009	341	12.46	26.42	48.62	64.93	71.74	35	51.4	56.26	45.34	37.77	36.37
n	0.35	10	10	10	10	10	10	10	10	10	10	10	10	10	10
SD	10	0.86	334.8	90.3	3.81	6.66	14.05	22.91	29.82	13.11	15.31	19.33	17.89	13.93	14.11
SEM	0.04	0.27	105.9	28.55	1.21	2.11	4.44	7.24	9.43	4.15	4.84	6.11	5.66	4.4	4.46

Appendix 10: Metamax Validity

SERVOMEX

	Heart Rate (bpm)				VO_2 (l.imn^{-1})				VCO_2 (l.imn^{-1})				Ventilation (l.min-1)				RER			
	Rest	1	2	3	Rest	1	2	3	Rest	1	2	3	Rest	1	2	3	Rest	1	2	3
Subject																				
1	78	112	141		0.31	0.94	1.44		0.26	0.72	1.2		8.68	22.54	33.38		0.83	0.77	0.83	
2		86	99	125		0.86	1.49	2.42		0.72	1.32	2.4		24.9	34.14	57.43		0.84	0.89	0.99
3	65	106	124	151	0.28	0.9	1.24	1.91	0.27	0.71	1.02	1.66	12.07	24.9	30.36	43.85	0.95	0.79	0.82	0.87
4	76	120	129	140	0.22	0.92	0.99	1.19	0.19	0.86	0.98	1.13	7.92	25.18	29.42	31.31	0.86	0.94	0.99	0.94
5	74	111	160		0.17	0.84	1.36		0.14	0.72	1.22		9.24	23.76	35.74		0.84	0.86	0.9	
average	73.25	107	130.6	138.7	0.25	0.89	1.3	1.84	0.21	0.75	1.15	1.73	9.48	24.25	32.61	44.2	0.87	0.84	0.89	0.93
n	4	5	5	3	4	5	5	3	4	5	5	3	4	5	5	3	4	5	5	3
SD	5.74	12.8	22.46	13.05	0.06	0.04	0.2	0.62	0.06	0.06	0.14	0.64	1.81	1.1	2.64	13.06	0.05	0.07	0.07	0.06
SEM	36.63	47.9	58.41	80.06	0.12	0.4	0.58	1.06	0.11	0.33	0.51	1	4.74	10.85	14.58	25.52	0.43	0.38	0.4	0.54

METAMAX

	Heart Rate (bpm)				VO_2 (l.imn^{-1})				VCO_2 (l.imn^{-1})				Ventilation (l.min-1)				RER			
	Rest	1	2	3	Rest	1	2	3	Rest	1	2	3	Rest	1	2	3	Rest	1	2	3
Subject																				
1	96	110	141		0.18	0.86	1.5		0.18	0.68	1.25		5.32	18.7	30.75		0.96	0.79	0.84	
2		79	95	118		1.11	1.64	2.44		0.95	1.48	2.35		25.46	38.17	57.6		0.85	0.91	0.96
3	62	106	123	150	0.12	0.58	0.83	1.08	0.09	0.5	0.79	1.07	9.57	21.46	38.04	54.07	0.76	0.87	0.95	0.99
4	76	123	129	131	0.59	1.57	1.61	2.08	0.47	1.35	1.43	1.76	11.4	31.44	34.33	40.28	0.79	0.86	0.88	0.85
5	73	111	158		0.25	0.74	1.12		0.17	0.53	0.9		4.8	13	20.4		0.66	0.72	0.8	
average	76.75	106	129.2	133	0.28	0.97	1.34	1.87	0.22	0.8	1.17	1.73	7.77	22.01	32.34	50.65	0.8	0.82	0.88	0.93
n	4	5	5	3	4	5	5	3	4	5	5	3	4	5	5	3	4	5	5	3
SD	14.17	16.3	23.33	16.09	0.21	0.39	0.35	0.7	0.16	0.35	0.31	0.64	3.23	6.95	7.34	9.15	0.12	0.06	0.06	0.08
SEM	7.09	7.28	10.43	9.29	0.1	0.17	0.16	0.41	0.08	0.16	0.14	0.37	1.61	3.11	3.28	5.29	0.06	0.03	0.03	0.04

Appendix 11: Incremental Exercise Test

	Peak Power (W)					Resting Heart Rate (bpm)					Peak Heart Rate (bpm)					Resting VO_2 ($L.min^{-1}$)				
Subject	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12
1	3.9	5	6.2	8.6	14.5	63	65	56	52	58	76	91	85	84	90	0.17	0.23	0.16	0.17	0.15
2	4.4	4.1	11.5	19	17	66	52	61	64	59		72	85	83	76	0.27	0.15	0.17	0.25	0.28
3	8.2	8.2	13.5	13	10	57	65	61	61	62	81	93	90	89	83	0.24	0.17	0.26	0.18	0.19
4	4	10	16	18.5	19	71	81	71	88	65	77	83	91	96	89	0.3	0.28	0.28	0.24	0.33
5	8.5	9.5	23	25.5	36.5	84	80	73	65	78	90	93	95	98	108	0.27	0.25	0.26	0.28	0.26
average	5.8	7.36	14.04	16.92	19.4	68.2	68.6	64.4	66	64.4	81	86.4	89.2	90	89.2	0.25	0.22	0.23	0.22	0.24
n	5	5	5	5	5	5	5	5	5	5	4	5	5	5	5	5	5	5	5	5
SD	2.34	2.67	6.17	6.42	10.13	10.18	12.1	7.27	13.32	8.08	6.38	9.04	4.27	6.82	11.9	0.05	0.05	0.06	0.05	0.07
SEM	1.05	1.19	2.76	2.87	4.53	4.55	5.41	3.25	5.96	3.61	3.19	4.04	1.91	3.05	5.32	0.02	0.02	0.03	0.02	0.03

	Peak VO_2 ($L.min^{-1}$)					Resting Ventilation ($L.min^{-1}$)					Peak Ventilation ($L.min^{-1}$)					Peak respiratory exchange ratio				
Subject	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12
1	0.24	0.43	0.46	0.49	0.87	4.72	6.63	4.44	5.55	5.87	9.09	15.52	15.26	15.31	24.4	0.92	1.13	1.2	0.99	1.13
2	0.57	0.24	0.76	0.85	0.77	5.48	4.18	4.83	6.91	6.55	12.82	6.68	20.08	21.91	20.7	0.84	0.91	0.92	1	0.97
3	0.5	0.53	0.58	0.5	0.54	5.57	4.28	6.08	4.31	5.08	13.19	17.66	17.58	15.12	19.25	1.02	1.19	1.07	1.14	1.28
4	0.44	0.46	0.73	0.69	0.69	8.33	8.39	6.77	6.34	7.92	11.14	13.71	19	20.21	15.59	0.81	1.13	1.05	1.05	1.13
5	0.53	0.6	0.71	0.82	0.92	8.03	8	9.23	7.49	8.68	19.35	19.94	23.09	26.46	30.95	1.26	1.09	1.1	1.08	1.14
average	0.46	0.45	0.65	0.67	0.76	6.43	6.3	6.27	6.12	6.82	13.12	14.7	19	19.8	22.18	0.97	1.09	1.07	1.05	1.13
n	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5
SD	0.13	0.14	0.13	0.17	0.15	1.64	2	1.9	1.24	1.47	3.84	5.05	2.91	4.77	5.83	0.18	0.11	0.1	0.06	0.11
SEM	0.06	0.06	0.06	0.08	0.07	0.73	0.89	0.85	0.55	0.66	1.72	2.26	1.3	2.13	2.61	0.08	0.05	0.05	0.03	0.05

	Peak [Lactate]					Anaerobic Threshold ($L.min^{-1}$)					Rates of perceived exertion					Rates of perceived breathlessness				
Subject	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12
1	5.8	5.1	5.8	3.9	7	0.18	0.29	0.31	0.3	0.29	12	12	10	7	12	3	3	3	2	3
2	2.8		8.7	14.5	5.87	0.33	0.17	0.38	0.34	0.4	9	9	11	11	9	1	2	2	3	1
3	3.3	5.7	4.8	4.5	4.27	0.13	0.21	0.19	0.18	0.2	9	13	12	12	12	1	3	3	3	2
4	4.1	4.4	6.1	5.5	5.28	0.21	0.24	0.36	0.33	0.36	9	8	9	8	9	0.5	0	0.5	0.5	2
5		5.7	6.4	6.33	9.55	0.36	0.34	0.34	0.43	0.39	15	9	13	7	13	4	1	4	0.5	3
average	4	5.23	6.36	6.95	6.39	0.24	0.25	0.32	0.32	0.33	10.8	10.2	11	9	11	1.9	1.8	2.5	1.8	2.2
n	4	4	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5	5
SD	1.31	0.62	1.44	4.32	2.02	0.1	0.07	0.08	0.09	0.08	2.68	2.17	1.58	2.35	1.87	1.52	1.3	1.32	1.25	0.84
SEM	0.66	0.31	0.64	1.93	0.9	0.04	0.03	0.03	0.04	0.04	1.2	0.97	0.71	1.05	0.84	0.68	0.58	0.59	0.56	0.37

Appendix 12: Constant Load Exercise Test

	Resting Heart Rate (bpm)					Steady State Heart Rate (bpm)					Resting VO_2 ($L.min^{-1}$)					Steady State VO_2 ($L.min^{-1}$)				
Subject	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12
1	65	85	62	77	49		110	81	94	69	0.22	0.22	0.17	0.18	0.21		0.43	0.35	0.32	0.34
2	65	55	60	69	66	64	62	71	71	67	0.32	0.22	0.17	0.23	0.28	0.45	0.4	0.33	0.36	0.43
3	72	68	64	60	68	79	82	71	70	72	0.17	0.24	0.19	0.15	0.17	0.43	0.51	0.32	0.35	0.32
4	75	75	68	66	74	82	81	74	70	73	0.35	0.39	0.26	0.29	0.27	0.46	0.47	0.34	0.43	0.34
5	68	83	75	70	78	84	96	88	76	96.3	0.28	0.26	0.27	0.27	0.28	0.51	0.48	0.4	0.44	0.38
average	69	73.2	65.8	68.4	67	77.25	86.2	77	76.2	75.46	0.27	0.27	0.21	0.22	0.24	0.46	0.46	0.35	0.38	0.36
n	5	5	5	5	5	4	5	5	5	5	5	5	5	5	5	4	5	5	5	5
SD	4.42	12.2	5.93	6.19	11.14	9.07	17.98	7.38	10.26	11.89	0.07	0.07	0.05	0.06	0.05	0.03	0.04	0.03	0.05	0.04
SEM	1.97	5.46	2.65	2.77	4.98	4.53	8.04	3.3	4.59	5.32	0.03	0.03	0.02	0.03	0.02	0.02	0.02	0.01	0.02	0.02

	Resting Ventilation ($l.min^{-1}$)					Steady State Ventilation ($L.min^{-1}$)					Respiratory exchange ratio					[Lactate]				
Subject	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12
1	7.75	6.01	4.96	5.11	6.42		17.95	12.23	9.59	10.76		1.19	1.09	0.86	0.88		10.7	5.87	4.42	4.17
2	6.69	5.54	5.01	6.13	7.54	11.09	11	9.3	9.94	11.74	0.82	0.88	0.88	0.86	0.83	4.8	2.5	5.96	11.9	7.82
3	4.21	6.27	4.3	4.67	4.45	13.71	16.61	9.43	10.54	10.84	1.06	1.04	0.93	0.99	1.07	8.2	7.91	2.99	4.19	5.3
4	9.01	9.75	7.36	7.74	7.67	13.85	13.49	9.12	11.27	9.06	0.92	0.98	0.86	0.86	0.91	5.2	4.15	2.49	4.43	3.96
5	8.01	8.39	10.32	8.45	8.76	17.75	16.2	14.25	13.08	12.06	1.09	1.04	0.98	0.89	0.94		3.98	3.55	2.7	2.58
average	7.13	7.19	6.39	6.42	6.97	14.1	15.05	10.87	10.88	10.89	0.97	1.03	0.95	0.89	0.93	6.07	5.85	4.17	5.53	4.77
n	5	5	5	5	5	4	5	5	5	5	4	5	5	5	5	3	5	5	5	5
SD	1.83	1.8	2.49	1.64	1.63	2.74	2.78	2.28	1.38	1.17	0.13	0.11	0.09	0.06	0.09	1.86	3.37	1.64	3.63	1.96
SEM	0.82	0.8	1.11	0.73	0.73	1.37	1.25	1.02	0.62	0.52	0.06	0.05	0.04	0.03	0.04	1.07	1.51	0.73	1.63	0.88

	Work Efficiency (%)					Stimulation Intensity (%)					Rates of perceived exertion					Rates of perceived breathlessness				
Subject	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12	0	3	6	9	12
1	3.11	3.11	4.6	6.15	7.94		100	66	65	14		12	10.7	7	7.7		3.5	2.7	1.2	1.2
2	4.67	6.13	6.77	8.01	5.7	34	28.5	19	9.5	11	7	7	7	7	7	0.5	0.5	0.7	0	0.7
3	6.6	6.38	15.04	9.46	12.83	82	67	26.5	33	28	11	12	9	10.3	11	1	2.7	1	1.3	2
4	9.85	9.84	19.15	11.88	17.15	100	42.5	10	6	14	9.3	7.3	7	6.7	7	0.5	0.2	0.3	0	0
5	6.26	6.98	11.53	14.66	17.53	68	53	28	15.5	18	10.3	6	6	6	6	0.8	0	0	0	0
average	6.1	6.49	11.42	10.03	12.23	71	58.2	29.9	25.8	17	9.4	8.86	7.94	7.4	7.74	0.7	1.38	0.94	0.5	0.78
n	5	5	5	5	5	4	5	5	5	5	4	5	5	5	5	4	5	5	5	5
SD	2.52	2.4	5.94	3.33	5.33	27.93	27.3	21.41	24.25	6.63	1.75	2.91	1.89	1.67	1.92	0.24	1.61	1.05	0.69	0.85
SEM	1.13	1.07	2.66	1.49	2.39	13.96	12.21	9.57	10.85	2.97	0.87	1.3	0.84	0.75	0.86	0.12	0.72	0.47	0.31	0.38

Appendix 13: T_cPO_2 Reliability

Experiment 1: Response to Loading

Subject/ Trial	TcPO2 (mmHg) Baseline	500g	700g	900g	1100g	1300g	1500g	Total weight (g)
1/1	80	42	5					700
1/2	69	54	50	44	21	5		1300
1/3	85	61	55	46	26	7		1300
1/4	67	53	51	49	33	19		1300
1/5	80	56	39	10				900
mean	**76.2**	**53.2**	**40**	**37.25**	**26.67**	**10.33**		**1100**
n	**5**	**5**	**5**	**4**	**3**	**3**		**5**
sd	**7.79**	**6.98**	**20.45**	**18.28**	**6.03**	**7.57**		**282.84**
sem	**3.48**	**3.12**	**9.14**	**9.14**	**3.48**	**4.37**		**126.49**
cv	**10.22**	**13.12**	**51.11**	**49.08**	**22.60**	**73.28**		**25.71**
2/1	57	61	53	40	1			1100
2/2	62	27	15					700
2/3	65	38	38	40	34	39	19	1500
2/4	62	34	16					700
2/5	64	28	10					700
mean	**62**	**37.6**	**26.4**	**40**	**17.5**	**39**	**19**	**940**
n	**5**	**5**	**5**	**2**	**2**	**1**	**1**	**5**
sd	**3.08**	**13.83**	**18.37**	**0.00**	**23.33**			**357.77**
sem	**1.38**	**6.19**	**8.21**	**0.00**	**16.50**			**160.00**
cv	**4.97**	**36.78**	**69.57**	**0.00**	**133.34**			**38.06**
3/1	54	31	27	15				900
3/2	65	46	47	40	31	9		1300
3/3	71	54	59	62	54	26	8	1500
3/4	69	24	17					700
3/5	61	46	37	26	4			1100
mean	**64**	**40.2**	**37.4**	**35.75**	**29.67**	**17.5**	**8**	**1100**
n	**5**	**5**	**5**	**4**	**3**	**2**	**1**	**5**
sd	**6.78**	**12.30**	**16.46**	**20.27**	**25.03**	**12.02**		**316.23**
sem	**3.03**	**5.50**	**7.36**	**10.14**	**14.45**	**8.50**		**141.42**
cv	**10.60**	**30.59**	**44.00**	**56.70**	**84.36**	**68.69**		**28.75**
4/1	63	49	47	38	36	9		1300
4/2	62	33	9					700
4/3	71	51	53	46	25	4		1300
4/4	68	60	56	42	12			1100
4/5	74	54	47	15				900
mean	**67.6**	**49.4**	**42.4**	**35.25**	**24.33**	**6.5**		**1060**
n	**5**	**5**	**5**	**4**	**3**	**2**		**5**
sd	**5.13**	**10.06**	**19.07**	**13.89**	**12.01**	**3.54**		**260.77**
sem	**2.29**	**4.50**	**8.53**	**6.94**	**6.94**	**2.50**		**116.62**
cv	**7.59**	**20.37**	**44.98**	**39.40**	**49.37**	**54.39**		**24.60**
5/1	73	49	43	30	14			1100
5/2	70	42	6					700
5/3	75	55	15					700
5/4	68	35	30	12				900
5/5	72	51	45	36	25	5		1300
mean	**71.6**	**46.4**	**27.8**	**26**	**19.5**	**5**		**940**
n	**5**	**5**	**5**	**3**	**2**	**1**		**5**
sd	**2.70**	**7.92**	**17.11**	**12.49**	**7.78**			**260.77**
sem	**1.21**	**3.54**	**7.65**	**7.21**	**5.50**			**116.62**
cv	**3.77**	**17.08**	**61.54**	**48.04**	**39.89**			**27.74**

Trial	1	2	3	4	5
		Subject 1			
Baseline	80	69	85	67	80
20	23			36	31
40	46	58	49	67	66
60	55	66	89	74	74
80	57	69	91	76	76
100	58	70	92	80	76
120	57	72	94	80	77
140	58	74	95	79	78
160	59	75	96	81	77
180	59	74	96	81	78
		Subject 3			
Baseline	54	65		71	
20				25	
40	40	43		48	
60	48	60		59	
80	50	62		64	
100	50	65		66	
120	52	65		67	
140	52	64		67	
160	51	65		66	
180	52	63		66	
		Subject 5			
Baseline	73	70	75	68	72
20	49	28	26	28	25
40	58	50	49	45	48
60	63	58	56	54	63
80	68	63	61	60	69
100	71	67	64	62	71
120	73	68	66	63	72
140	74	67	65	64	72
160	76	68	66	65	72
180	77	68	66	66	73

Trial	1	2	3	4	5
		Subject 2			
Baselin	57	62	65	74	64
20		71		31	12
40	74	69	49	44	22
60	80	66	65	50	27
80	81	66	70	53	29
100	79	65	72	53	33
120	77	65	73	54	34
140	78	65	74	49	37
160	78	64	75	51	41
180	78	63	75	53	44
		Subject 4			
Baselin	63	65	71	68	74
20	27		32	16	30
40	51	53	53	25	56
60	60	64	62	33	62
80	64	68	66	38	67
100	66	69	65	42	68
120	66	71	69	47	69
140	66	72	70	50	70
160	68	73	69	53	67
180	68	73	72	56	68

Trial	1	2	3	4	5
		Subject 1			
Baseline	83	78	79	75	81
20	32	28	20	20	27
40	58	51	42	45	50
60	68	62	52	61	65
80	74	68	62	66	70
100	83	72	68	71	75
120	84	72	72	73	75
140	83	79	74	75	78
160	83	81	76	75	80
180	84	81	78	75	80
		Subject 3			
Baseline	78	70	75	68	72
20	26	3	9	5	12
40	34	25	35	28	37
60	46	45	56	45	57
80	58	54	65	54	67
100	65	59	71	60	70
120	70	62	75	62	71
140	73	65	76	63	72
160	75	65	76	64	72
180	76	66	77	65	73
		Subject 5			
Baseline	77	79	78	87	72
20	5	16	7	10	5
40	25	28	18	21	15
60	44	40	38	45	30
80	60	57	59	68	54
100	67	67	69	79	65
120	70	74	73	83	70
140	73	79	76	86	71
160	75	80	75	88	71
180	75	80	75	90	72

Trial	1	2	3	4	5
		Subject 2			
Baselin	59	67	70	55	62
20	4	9	5	11	5
40	15	23	18	29	23
60	30	39	34	40	32
80	42	51	45	46	41
100	52	60	54	51	47
120	56	63	61	53	51
140	58	62	65	54	54
160	60	66	68	55	57
180	60	67	70	55	58
		Subject 4			
Baselin	78	86	81	81	80
20	25	15	21	14	22
40	57	52	50	50	51
60	68	63	58	66	62
80	73	72	65	73	69
100	75	77	72	77	73
120	77	77	75	78	75
140	77	77	77	79	75
160	76	76	79	77	74
180	76	77	79	76	74

Appendix 14: Tissue Oxygenation

Subject	1	2	3	4	Mean	SD	n	SEM	1	2	3	4	Mean	SD	n	SEM
Time (s)	Baseline Recovery (mmHg)								3 Month Recovery (mmHg)							
Baseline	60	67	50	97	68.5	20.24	4	10.12	94	74	59	56	70.75	17.39	4	8.6927
20	51	17	9		25.667	22.301	3	12.875	31	5	76	8	30	32.79	4	16.396
40	69	39	18		42	25.632	3	14.799	66	50	81	25	55.5	23.95	4	11.976
60	73	49	29		50.333	22.03	3	12.719	76	60	79	39	63.5	18.34	4	9.1697
80	75	56	38		56.333	18.502	3	10.682	82	66	75	47	67.5	15.15	4	7.5774
100	74	60	45		59.667	14.503	3	8.3732	83	70	72	51	69	13.29	4	6.6458
120	73	63	49		61.667	12.055	3	6.9602	83	72	69	53	69.25	12.39	4	6.1964
140	76	65	52		64.333	12.014	3	6.9362	84	73	67	56	70	11.69	4	5.8452
160	79	66	53		66	13	3	7.5056	85	75	66	56	70.5	12.4	4	6.1981
180	82	68	54		68	14	3	8.0829	84	76	66	57	70.75	11.76	4	5.879
	6 Month Recovery (mmHg)								9 Month Recovery (mmHg)							
Baseline	74	77	59	55	66.25	10.874	4	5.4371	58	71	53	59	60.25	7.632	4	3.8161
20	25	17	28	5	18.75	10.275	4	5.1377	12	27	11	13	15.75	7.544	4	3.7722
40	52	42	43	20	39.25	13.598	4	6.7992	33	49	33	32	36.75	8.18	4	4.0901
60	62	54	45	33	48.5	12.45	4	6.2249	46	63	50	45	51	8.287	4	4.1433
80	67	59	49	45	55	9.9331	4	4.9666	53	70	57	53	58.25	8.057	4	4.0285
100	69	63	53	50	58.75	8.8081	4	4.4041	58	75	61	59	63.25	7.932	4	3.966
120	71	64	56	52	60.75	8.4607	4	4.2303	60	79	63	60	65.5	9.11	4	4.5552
140	71	67	58	54	62.5	7.8528	4	3.9264	61	81	63	61	66.5	9.713	4	4.8563
160	71	69	59	54	63.25	8.0984	4	4.0492	61	81	63	62	66.75	9.535	4	4.7675
180	72	72	60	54	64.5	9	4	4.5	61	80	64	63	67	8.756	4	4.378
	12 Month Recovery (mmHg)															
Baseline	67	58	80	50	63.75	12.868	4	6.434								
20	14	28	30	3	18.75	12.685	4	6.3426								
40	40	45	64	7	39	23.707	4	11.853								
60	57	48	73	21	49.75	21.777	4	10.889								
80	62	51	78	35	56.5	18.12	4	9.06								
100	64	53	81	44	60.5	15.927	4	7.9635								
120	66	55	83	48	63	15.253	4	7.6267								
140	67	57	84	48	64	15.427	4	7.7136								
160	68	59	84	48	64.75	15.218	4	7.6089								
180	67	60	83	48	64.5	14.617	4	7.3087								

Appendix 15: Seating Pressure

NHS Wheelchair

Subject		Left										Left Max	Left Average	Left SD
Baseline		**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**			
	1	93	59	60	74	89	61	76	71	75	71	93	72.9	11.483805
	2	60	57	63	57	58	60	59	57	58	60	63	58.9	1.9119507
	3	88	99	77	98	105	73	70	67	60	63	105	80	16.295875
	4	100	75	89	93	85	100	107	91	70	62	107	87.2	14.358892
	5	42	44	47	47	42	47	38	45	48	41	48	44.1	3.2812599
Average		**76.6**	**66.8**	**67.2**	**73.8**	**75.8**	**68.2**	**70**	**66.2**	**62.2**	**59.4**	**83.2**	**68.62**	**9.4663566**
SD		**24.5926818**	**21.1**	**16.2**	**22.107**	**25.35**	**20.017**	**25.2**	**17.1**	**10.59245**	**11.10405**		**19.342897**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**10.9981817**	**9.4361**	**7.24**	**9.8864**	**11.34**	**8.9521**	**11.3**	**7.66**	**4.737088**	**4.965884**		**8.6504064**	
3 Month														
	1	50	51	51	47	49	49	54	59	56		59	51.777778	3.8333333
	2	69	56	52	52	47	41	50	41	46	49	69	50.3	8.111035
	3	75	85	73	66	65	58	67	68	68	63	85	68.8	7.4206918
	4	113	82	105	90	96	112	67	97	92	93	113	94.7	13.776389
	5	44	40	40	46	49	48	46	45	45	52	52	45.5	3.7193189
Average		**70.2**	**62.8**	**64.2**	**60.2**	**61.2**	**61.6**	**56.8**	**62**	**61.4**	**64.25**	**75.6**	**62.215556**	**7.3721536**
SD		**27.1606333**	**19.791**	**25.7**	**18.472**	**20.77**	**28.815**	**9.73**	**22.4**	**19.46279**	**20.08938**		**21.239113**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**4**		**4.9**	
SEM		**12.1466045**	**8.851**	**11.5**	**8.2608**	**9.287**	**12.886**	**4.35**	**10**	**8.704022**	**10.04469**		**9.6044647**	
6 Month														
	1	56	48	47	46	48	57	77	52	51	49	77	53.1	9.1706052
	2	76	71	69	68	67	67	67	64	65	65	76	67.9	3.5103023
	3	118	124	100	103	88	87	79	71	88	94	124	95.2	16.511276
	4	71	62	77	120	66	100	102	70	107	79	120	85.4	20.089798
	5	60	55	49	46	50	57	52	48	50	47	60	51.4	4.575296
Average		**76.2**	**72**	**68.4**	**76.6**	**63.8**	**73.6**	**75.4**	**61**	**72.2**	**66.8**	**91.4**	**70.6**	**10.771456**
SD		**24.7224594**	**30.29**	**21.8**	**33.642**	**16.13**	**19.178**	**18.3**	**10.5**	**24.77297**	**20.005**		**21.937679**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**11.05622**	**13.546**	**9.77**	**15.045**	**7.214**	**8.5767**	**8.19**	**4.69**	**11.07881**	**8.946508**		**9.8108284**	

Appendix 15: Seating Pressure

NHS Wheelchair

9 months														
	1	69	69	67	80	86	89	76	69	58	55	89	71.8	11.063453
	2	60	57	54	55	58	55	51	56	60	60	60	56.6	2.9888682
	3	104	114	117	117	141	112	132	100	129	113	141	117.9	12.670613
	4	78	85	64	68	69	69	63	84	88	90	90	75.8	10.347302
	5	52	60	50	61	54	53	47	50	52	53	61	53.2	4.3410188
Average		**72.6**	**77**	**70.4**	**76.2**	**81.6**	**75.6**	**73.8**	**71.8**	**77.4**	**74.2**	**88.2**	**64.2**	**5.7095939**
SD		**20.0698779**	**23.377**	**27**	**24.631**	**35.44**	**24.916**	**34.4**	**20.5**	**32.0125**	**26.33819**		**26.868075**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**8.97552227**	**10.455**	**12.1**	**11.015**	**15.85**	**11.143**	**15.4**	**9.16**	**14.31642**	**11.77879**		**12.015768**	
12 months														
	1	86	62	71	93	86	89	102	102	100	81	102	87.2	13.239671
	2	100	97	93	93	91	82	94	107	107	100	107	96.4	7.5748121
	3	112	100	100	100	92	92	78	85	92	75	112	92.6	11.167413
	4	106	109	97	144	121	119	146	170	156	100	170	126.8	25.459336
	5	60	79	77	69	62	57	63	71	69	66	79	67.3	7.1344236
Average		92.8	89.4	87.6	99.8	90.4	87.8	96.6	107	104.8	84.4	114	91.8	4.0056602
SD		20.7171427	**18.796**	**12.8**	**27.362**	**21.01**	**22.219**	**31.4**	**38**	**31.99531**	**15.20855**		**23.956632**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**9.26498786**	**8.406**	**5.74**	**12.237**	**9.395**	**9.9368**	**14.1**	**17**	**14.30874**	**6.80147**		**10.713731**	

Subject		Right										Right Max	Right Average	Right SD
Baseline		1	2	3	4	5	6	7	8	9	10			
	1	99	114	107	77	82	75	76	84	61	78	114	85.3	16.33027
	2	53	52	50	54	55	54	55	53	57	56	57	53.9	2.024846
	3	80	74	79	89	84	88	78	80	71	81	89	80.4	5.601587
	4	114	103	116	121	132	140	169	125	133	112	169	126.5	18.62644
	5	45	54	54	56	56	55	56	59	60	55	60	55	4.027682
Average		**78.2**	**79.4**	**81.2**	**79.4**	**81.8**	**82.4**	**86.8**	**80.2**	**76.4**	**76.4**	**97.8**	**80.22**	**9.322166**
SD		**29.38877337**	**28.192**	**30**	**27.483**	**31.26**	**35.218**	**47.2**	**28.3**	**32.074912**	**23.26585**		**31.2391355**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**13.143059**	**12.608**	**13.4**	**12.291**	**13.98**	**15.75**	**21.1**	**12.7**	**14.344337**	**10.40481**		**13.9705661**	
3 Month														
	1	111	96	125	122	92	92	86	81	95		125	100	15.6365
	2	66	74	75	65	59	51	68	63	64	62	75	64.7	6.961002
	3	139	104	113	118	107	125	79	87	89	107	71	106.8	18.32303
	4	112	142	105	106	110	89	118	119	106	110	142	111.7	13.50761
	5	63	57	68	67	72	72	73	66	64	68	73	67	4.876246
Average		**98.2**	**94.6**	**97.2**	**95.6**	**88**	**85.8**	**84.8**	**83.2**	**83.6**	**86.75**	**97.2**	**90.04**	**11.86088**
SD		**32.76736181**	**32.308**	**24.6**	**27.664**	**22.12**	**27.326**	**19.7**	**22.4**	**18.902381**	**25.26361**		**25.3125507**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**4**		**4.9**	
SEM		**14.65400969**	**14.449**	**11**	**12.372**	**9.894**	**12.22**	**8.83**	**10**	**8.4534017**	**12.63181**		**11.4534743**	
6 Month														
	1	65	52	55	56	60	63	53	50	54	52	65	56	5.033223
	2	70	69	69	64	67	69	65	69	67	65	70	67.4	2.1187
	3	161	110	113	141	113	135	115	87	94	94	161	116.3	23.27158
	4	123	123	127	97	121	95	107	107	90	105	127	109.5	13.26022
	5	52	45	46	47	47	44	45	44	44	46	52	46	2.403701
Average		**94.2**	**79.8**	**82**	**81**	**81.6**	**81.2**	**77**	**71.4**	**69.8**	**72.4**	**95**	**79.04**	**9.217485**
SD		**46.1269986**	**34.924**	**36**	**38.49**	**33.22**	**35.174**	**32**	**26.1**	**21.890637**	**25.96729**		**32.9853292**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**20.62862089**	**15.619**	**16.1**	**17.213**	**14.86**	**15.73**	**14.3**	**11.7**	**9.7897906**	**11.61292**		**14.7514877**	

9 months														
	1	69	59	55	55	59	53	52	62	59	56	69	57.9	4.976612
	2	58	59	60	60	54	55	55	52	56	62	62	57.1	3.17805
	3	152	141	148	143	132	136	112	135	125	130	152	135.4	11.64474
	4	113	115	100	100	105	90	85	95	75	79	115	95.7	13.54047
	5	69	64	67	63	64	63	67	68	70	70	70	66.5	2.798809
Average		**92.2**	**87.6**	**86**	**84.2**	**82.8**	**79.4**	**74.2**	**82.4**	**77**	**79.4**	**82.8666667**	**82.52**	**1.271776**
SD		**39.54364677**	**38.063**	**38.9**	**37.399**	**34.14**	**34.918**	**24.8**	**33.4**	**27.93743**	**29.57702**		**33.8667645**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**17.68445645**	**17.022**	**17.4**	**16.725**	**15.27**	**15.616**	**11.1**	**15**	**12.493999**	**13.22724**		**15.1456775**	
12 months														
	1	62	64	64	63	77	66	81	63	83	65	83	68.8	8.162244
	2	86	74	75	83	83	75	67	106	125	125	125	89.9	21.22603
	3	124	141	159	117	96	96	111	100	119	107	159	117	20.27588
	4	103	115	94	133	103	94	106	115	94	84	115	104.1	14.09846
	5	60	71	80	67	65	62	53	68	62	66	80	65.4	7.152311
Average		**87**	**93**	**94.4**	**92.6**	**84.8**	**78.6**	**83.6**	**90.4**	**96.6**	**89.4**	**89**	**89.04**	**9.237493**
SD		**27.29468813**	**33.444**	**37.7**	**31.029**	**15.11**	**15.71**	**24.9**	**23.4**	**25.967287**	**26.21641**		**26.0725801**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**12.20655562**	**14.957**	**16.9**	**13.877**	**6.756**	**7.0257**	**11.1**	**10.5**	**11.612924**	**11.72433**		**11.6600123**	

Subject													Left Max	Left Average	Left SD
Baseline		**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**				
	1	45	63	49	50	46	54	47	48	49	56		63	50.7	5.498485
	2	62	65	62	69	61	76	70	68	65	67		76	66.5	4.552167
	3	114	116	110	118	120	116	109	105	93	94		120	109.5	9.548124
	4	63	53	49	61	43	35	37	45	38	34		63	45.8	10.49656
	5	48	49	49	50	50	51	52	52	50	54		54	50.5	1.779513
Average		**66.4**	**69.2**	**63.8**	**69.6**	**64**	**66.4**	**63**	**63.6**	**59**	**61**		**75.2**	**64.6**	**6.37497**
SD		**27.8**	**27.004**	**26.4**	**28.219**	**32.04**	**31.342**	**28.4**	**24.8**	**21.29554**	**21.9545**			**26.92432251**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**			**5**	
SEM		**12.4**	**12.076**	**11.8**	**12.62**	**14.33**	**14.016**	**12.7**	**11.1**	**9.523655**	**9.81835**			**12.04092308**	
3 Month															
	1	53	49	53	50	42	46	42					53	47.85714286	4.670067
	2	52	50	53	54	55	51	54	51	53	55		55	52.8	1.75119
	3	79	71	70	64	69	69	65	70	64	66		79	68.7	4.473378
	4	51	52	55	67	65	71	38	45	50			67	54.88888889	10.83333
	5	52	51	51	49	54	55	51	51	44	49		55	50.7	3.020302
Average		**57.4**	**54.6**	**56.4**	**56.8**	**57**	**58.4**	**50**	**54.3**	**52.75**	**56.66667**		**61.8**	**55.28888889**	**4.949654**
SD		**12.1**	**9.2358**	**7.73**	**8.228**	**10.56**	**11.082**	**10.6**	**10.9**	**8.381527**	**8.621678**			**9.866175927**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**4**	**4**	**3**			**4.777777778**	
SEM		**5.41**	**4.1304**	**3.46**	**3.6797**	**4.722**	**4.9558**	**4.74**	**5.44**	**4.190764**	**4.977728**			**4.525226334**	
6 Month															
	1	50	51	49	52	53	52	52	46	48	49		53	50.2	2.20101
	2	70	70	70	69	69	75	77	71	68	84		84	72.3	4.98999
	3	79	87	76	88	82	94	91	94	85	85		94	86.1	6.008328
	4	28	28	28	28	27	28	25	26	28	27			27.3	1.05935
	5	46	45	41	46	40	48	42	40	43	42		48	43.3	2.790858
Average		**54.6**	**56.2**	**52.8**	**56.6**	**54.2**	**59.4**	**57.4**	**55.4**	**54.4**	**57.4**		**69.75**	**55.84**	**3.409907**
SD		**20.2**	**22.841**	**20**	**22.865**	**21.99**	**25.55**	**26.6**	**27**	**22.30022**	**25.98653**			**23.54001848**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**			**5**	
SEM		**9.04**	**10.215**	**8.95**	**10.225**	**9.836**	**11.426**	**11.9**	**12.1**	**9.972963**	**11.62153**			**10.5274163**	

Own Wheelchair

9 months														
	1	49	49	46	46	44	43	41	46	42	44	49	45	2.708013
	2	71	66	64	72	72	73	69	70	66	68	73	69.1	3.034981
	3	112	113	104	100	121	100	89	104	92	96	113	103.1	9.949316
	4	86	66	80	90	95	93	79	85	88	86	95	84.66666667	8.803408
	5		73	52	51	50	48	48	51	50	48	73	52.33333333	7.889867
Average		**79.5**	**73.4**	**69.2**	**71.8**	**76.4**	**71.4**	**65.2**	**71.2**	**67.6**	**68.4**	**80.6**	**71.41**	**6.477117**
SD		**26.5**	**23.839**	**23.4**	**23.584**	**32.02**	**25.696**	**20.3**	**24**	**22.2441**	**22.82104**		**24.44235193**	
n		**4**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**4.9**	
SEM		**13.2**	**10.661**	**10.5**	**10.547**	**14.32**	**11.492**	**9.09**	**10.7**	**9.947864**	**10.20588**		**11.07064503**	
12 months														
	1	54	59	54	54	51	59	57	63	57	53	63	56.1	3.573047
	2		100	100	96	83	84	80	90	90	87	100	90	7.331439
	3	147	126	150	135	124	120	120	161	150	139	150	137.2	14.48984
	4	68	70	64	67	65	72	69	60	60	60	72	65.5	4.428443
	5	36	32	38	38	37	35	37	35	34	37	38	35.9	1.911951
Average		**76.3**	**77.4**	**81.2**	**78**	**72**	**74**	**72.6**	**81.8**	**78.2**	**75.2**	**84.6**	**76.665**	**6.346945**
SD		**49**	**36.494**	**44.7**	**38.308**	**33.69**	**31.488**	**30.9**	**48.4**	**44.80179**	**39.97749**		**39.76950275**	
n		**4**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**4.9**	
SEM		**24.5**	**16.321**	**20**	**17.132**	**15.07**	**14.082**	**13.8**	**21.6**	**20.03597**	**17.87848**		**18.04385972**	

Subject		Right										Right Max	Right Average	Right SD
Baseline		**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**			
	1	56	49	42	51	52	50	46	53	51	53	56	50.3	3.945462
	2	104	96	81	75	93	76	76	70	81	71	104	82.3	11.48961
	3	76	101	89	88	81	73	79	75	80	85	101	82.7	8.367264
	4	57	50	42	49	40	51	45	51	58	59	59	50.2	6.545567
	5	48	47	49	45	49	53	53	56	58	52	58	51	4.109609
Average		**68.2**	**68.6**	**60.6**	**61.6**	**63**	**60.6**	**59.8**	**61**	**65.6**	**64**	**75.6**	**63.3**	**6.891502**
SD		**22.5**	**27.373**	**22.6**	**18.863**	**22.75**	**12.779**	**16.5**	**10.8**	**13.90324**	**13.96424**		**18.204077**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**10.1**	**12.242**	**10.1**	**8.4356**	**10.17**	**5.7149**	**7.37**	**4.83**	**6.217717**	**6.244998**		**8.1411109**	
3 Month														
	1	49	47	49	47	51	46	44				49	47.571429	2.299068
	2	83	73	76	72	74	69	72	70	68	71	83	72.8	4.289522
	3	102	80	86	78	82	81	91	81	67	67	102	81.5	10.36286
	4	42	43	49	55	45	50	47	46	42		55	46.555556	4.275252
	5	100	71	78	81	74	77	70	81	83	83	100	79.8	8.495751
Average		**75.2**	**62.8**	**67.6**	**66.6**	**65.2**	**64.6**	**64.8**	**69.5**	**65**	**73.66667**	**77.8**	**67.496667**	**5.944491**
SD		**28.2**	**16.649**	**17.4**	**14.876**	**16.18**	**15.821**	**19.5**	**16.5**	**16.99019**	**8.326664**		**17.039803**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**4**	**4**	**3**		**4.6**	
SEM		**12.6**	**7.4458**	**7.78**	**6.6528**	**7.235**	**7.0753**	**8.7**	**8.25**	**8.495097**	**4.807402**		**7.9055878**	
6 Month														
	1	47	48	54	53	55	45	46	54	52	57	36	51.1	4.228212
	2	83	81	82	87	87	91	86	84	89	72	89	84.2	5.308274
	3	111	87	88	85	82	73	79	73	79	76	111	83.3	11.10605
	4	35	37	37	36	34	35	38	35	36	37	37	36	1.247219
	5	97	74	62	64	97	82	80	85	78	83	97	80.2	11.71703
Average		**74.6**	**65.4**	**64.6**	**65**	**71**	**65.2**	**65.8**	**66.2**	**66.8**	**65**	**74**	**66.96**	**6.721359**
SD		**32.5**	**21.755**	**20.8**	**21.622**	**25.87**	**24.129**	**22.1**	**21.4**	**21.99318**	**18.31666**		**23.05161**	
n		**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**5**	
SEM		**14.5**	**9.7293**	**9.3**	**9.6695**	**11.57**	**10.791**	**9.87**	**9.59**	**9.835649**	**8.191459**		**10.308993**	

9 months														
	1	51	51	51	51	49	51	53	46	53	49	53	50.5	2.068279
	2	63	60	58	63	68	73	63	62	76	60	76	64.6	5.891614
	3	100	88	84	88	100	84	89	92	100	81	100	90.6	7.167829
	4	86	85	73	60	58	56	56	54	54	51	86	60.777778	11.05416
	5		70	74	74	76	82	76	57	69	76	82	72.666667	6.98212
Average		**75**	**70.8**	**68**	**67.2**	**70.2**	**69.2**	**67.4**	**62.2**	**70.4**	**63.4**	**79.4**	**68.38**	**6.6328**
SD		**22.1**	**15.865**	**13.3**	**14.237**	**19.52**	**15.023**	**15**	**17.6**	**19.24318**	**14.50172**		**16.640354**	
n		**4**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**4.9**	
SEM		**11.1**	**7.0951**	**5.94**	**6.3671**	**8.732**	**6.7186**	**6.7**	**7.89**	**8.605812**	**6.485368**		**7.5584812**	
12 months														
	1	51	51	52	50	47	46	47	46	49	49	52	48.8	2.20101
	2		92	100	96	97	90	97	93	97	93	100	95	3.162278
	3	84	111	125	105	95	100	100	133	135	117	135	110.5	16.84076
	4	61	59	50	49	49	55	55	56	55	55	61	54.4	4.033196
	5	79	62	62	60	61	67	60	67	60	57	79	63.5	6.276057
Average		**68.8**	**75**	**77.8**	**72**	**69.8**	**71.6**	**71.8**	**79**	**79.2**	**74.2**	**85.4**	**73.915**	**6.502659**
SD		**15.4**	**25.426**	**33.2**	**26.561**	**24.52**	**22.898**	**24.8**	**34.9**	**36.38956**	**29.5161**		**27.366385**	
n		**4**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**	**5**		**4.9**	
SEM		**7.71**	**11.371**	**14.8**	**11.879**	**10.97**	**10.24**	**11.1**	**15.6**	**16.27391**	**13.2**		**12.319983**	

Appendix 16: Cardiopulmonary Responses to FES Cycling

Peak Power Test

Timepoint				Power									HR						
Subject	1	2	3	4	5	Average	n	SD	SEM	1	2	3	4	5	Average	% peak	n	SD	SEM
Rest										54	59	54		85	63	70.6278	4	14.85	7.427
Passive	-1.05	-4.18	-3.8	-1.42	-3.91	-2.87278	5	1.508	0.67	54	58	49		63	56	62.7803	4	5.944	2.972
1	22.76	26.78	29	29.5	60.4	33.77412	5	15.15	6.78	56	60	63		87	66.5	74.5516	4	13.96	6.982
2	10.25	11.15	8	20.3	31.2	16.18588	5	9.642	4.31	77	75	76		103	82.75	92.7691	4	13.52	6.762
3	7.444	7.008	4.5	12.1	26.6	11.51765	5	8.856	3.96	97	74	75		104	87.5	98.0942	4	15.29	7.643
4	9.069	7.603	4.5	11	27	11.82309	5	8.826	3.95	94	68	73		103	84.5	94.7309	4	16.7	8.352
5	9.851	9.315	4.8	11.7	26	12.32561	5	8.044	3.6	93	72	75		106	86.5	96.9731	4	15.97	7.984
6	10.85	10.52	5	12.3	26.3	12.98172	5	7.938	3.55	94	71	73		100	84.5	94.7309	4	14.66	7.331
7	11.28	11.19	4.8	12.7	26.3	13.26834	5	7.91	3.54	93	76	74		100	85.75	96.1323	4	12.76	6.382
8	11.52	11.46	5	12.7	24.5	13.05249	5	7.084	3.17	96	77	76		103	88	98.6547	4	13.59	6.795
9	11.97	11.8	5.1	13.2	28.6	14.1374	5	8.662	3.87	94	75	73		104	86.5	96.9731	4	15.02	7.511
10	12.09	11.81	5	13.5	27.8	14.03261	5	8.381	3.75	99	75	73		104	87.75	98.3744	4	16.03	8.014

	RER									VO2									
	1	2	3	4	5	Average	n	SD	SEM	1	2	3	4	5	Average	% peak	n	SD	SEM
Rest	0.681	0.676	0.6	0.75	0.81	0.707995	5	0.071	0.03	0.193	0.26	0.18348	0.31173	0.243	0.238641	31.483	5	0.052	0.023
Passive	0.743	0.777	0.7	0.76	0.89	0.773994	5	0.072	0.03	0.236	0.27	0.23624	0.3658	0.218	0.265367	35.0089	5	0.059	0.027
1	0.683	0.761	0.8	0.73	0.84	0.757348	5	0.057	0.03	0.32	0.35	0.2862	0.5848	0.524	0.413179	54.5091	5	0.133	0.059
2	0.996	0.934	0.9	1.01	1.17	1.009409	5	0.098	0.04	0.649	0.84	0.54627	0.76675	0.871	0.733795	96.8067	5	0.135	0.06
3	1.334	1.145	1.2	1.25	1.29	1.246778	5	0.072	0.03	0.719	0.82	0.50969	0.63837	0.853	0.70848	93.467	5	0.14	0.063
4	1.306	1.119	1.3	1.25	1.28	1.259013	5	0.086	0.04	0.701	0.73	0.51069	0.67333	0.87	0.698048	92.0908	5	0.129	0.058
5	1.243	1.064	1.3	1.17	1.23	1.207586	5	0.101	0.05	0.737	0.8	0.52194	0.56655	0.863	0.698238	92.1158	5	0.148	0.066
6	1.211	1.024	1.4	1.13	1.2	1.181539	5	0.12	0.05	0.728	0.8	0.54033	0.64545	0.876	0.717404	94.6444	5	0.131	0.058
7	1.166	0.994	1.3	1.14	1.16	1.144221	5	0.096	0.04	0.738	0.82	0.50924	0.59689	0.869	0.707559	93.3455	5	0.152	0.068
8	1.186	1.003	1.2	1.13	1.15	1.138404	5	0.083	0.04	0.73	0.82	0.50907	0.61665	0.856	0.707206	93.299	5	0.145	0.065
9	1.159	0.979	1.2	1.14	1.08	1.104305	5	0.078	0.04	0.742	0.89	0.50513	0.59845	0.895	0.726721	95.8735	5	0.174	0.078
10	1.147	0.994	1.2	1.1	1.12	1.106869	5	0.069	0.03	0.755	0.85	0.55094	0.607	0.896	0.732093	96.5822	5	0.15	0.067

Appendix 16: Cardiopulmonary Responses to FES Cycling

Peak Power Test

	VCO2									V									
	1	2	3	4	5	Average	n	SD	SEM	1	2	3	4	5	Average	% peak	n	SD	SEM
Rest	0.132	0.178	0.1	0.23	0.2	**0.170804**	**5**	**0.048**	**0.02**	5.63	6.1	4.26	7.53	7.22	**6.148**	**27.7212**	**5**	**1.313**	**0.587**
Passive	0.175	0.21	0.2	0.28	0.19	**0.204381**	**5**	**0.044**	**0.02**	7.066	8.07	6.34704	8.97478	10.88	**8.267506**	**37.278**	**5**	**1.767**	**0.79**
1	0.216	0.268	0.2	0.42	0.42	**0.309583**	**5**	**0.105**	**0.05**	8.087	9.35	7.89681	11.0228	12.14	**9.699387**	**43.7343**	**5**	**1.85**	**0.827**
2	0.651	0.79	0.5	0.77	1.02	**0.748483**	**5**	**0.187**	**0.08**	16.9	19.6	14.0343	18.9718	25.46	**18.9973**	**85.6583**	**5**	**4.218**	**1.886**
3	0.958	0.94	0.6	0.79	1.1	**0.883078**	**5**	**0.183**	**0.08**	23.84	22.9	16.9577	19.964	28.99	**22.53025**	**101.588**	**5**	**4.506**	**2.015**
4	0.916	0.823	0.7	0.84	1.11	**0.875542**	**5**	**0.156**	**0.07**	25.08	20.9	18.1388	21.4616	30.48	**23.21805**	**104.69**	**5**	**4.754**	**2.126**
5	0.914	0.855	0.7	0.66	1.06	**0.837232**	**5**	**0.162**	**0.07**	26.57	22.3	20.9702	17.878	29.36	**23.40985**	**105.554**	**5**	**4.56**	**2.039**
6	0.882	0.816	0.7	0.73	1.05	**0.839773**	**5**	**0.133**	**0.06**	26.95	22	21.1064	19.2728	29.91	**23.85495**	**107.561**	**5**	**4.419**	**1.976**
7	0.861	0.819	0.6	0.68	1.01	**0.802701**	**5**	**0.148**	**0.07**	26.78	22	19.4735	18.2831	30.14	**23.33877**	**105.234**	**5**	**5.009**	**2.24**
8	0.865	0.828	0.6	0.7	0.98	**0.799194**	**5**	**0.141**	**0.06**	28.04	22.7	19.0648	18.8013	30.38	**23.7931**	**107.282**	**5**	**5.242**	**2.344**
9	0.86	0.876	0.6	0.68	0.96	**0.794388**	**5**	**0.153**	**0.07**	28.19	23.8	18.9668	19.0646	29.58	**23.91626**	**107.838**	**5**	**4.959**	**2.218**
10	0.866	0.847	0.6	0.67	1	**0.805903**	**5**	**0.147**	**0.07**	29.05	23.1	20.0645	18.6475	31.04	**24.37262**	**109.896**	**5**	**5.464**	**2.444**

Appendix 16: Cardiopulmonary Responses to FES Cycling

Home Training Data

	Power					HR					RER				
timepoint	1	2	3	4	Average	1	2	3	4	Average	1	2	3	4	Average
Rest						64	59	64	97	71	0.812	0.86	0.81	0.889	0.84437
1	14	10	13	18	13.75						0.809	0.91	0.84	0.867	0.854877
2											0.805	1.07	0.98	1.175	1.008268
3											0.875	1.38	1.26	1.37	1.222136
4											0.929	1.52	1.35	1.362	1.290411
5	9	4	12	15	10	76	72	75	98	80.25	1.122	1.46	1.17	1.286	1.258587
6											1.126	1.49	1.22	1.207	1.262123
7											1.082	1.3	1.19	1.179	1.187763
8											1.036	1.29	1.15	1.144	1.154331
9											1.023	1.52	1.12	1.13	1.199748
10	8	5	8	16	9.25	73	83	82	102	85	1.042	1.42	1.11	1.095	1.167649
11											1.028	1.36	1.13	1.091	1.153622
12											1.008	1.26	1.11	1.109	1.120696
13											1.005	1.27	1.1	1.085	1.113733
14											0.982	1.24	1.12	1.122	1.115715
15	8	5	7	15	8.75	75	79	88	108	87.5	0.973	1.25	1.12	1.079	1.103337
16											0.966	1.41	1.08	1.093	1.13865
17											0.969	1.46	1.09	1.085	1.151661
18											0.955	1.49	1.08	1.069	1.14978
19											0.982	1.25	1.08	1.051	1.091398
20	7	5	8	14	8.5	77	78	91	112	89.5	0.961	1.11	1.07	1.07	1.055029
21											0.952	1.18	1.06	1.055	1.062624
22											0.938	1.22	1.06	1.044	1.064913
23											0.947	1.07	1.06	1.063	1.034235
24											0.943	1.09	1.06	1.028	1.029661
25	5	3	8	12	7	74	83	94	104	88.75	0.929	0.99	1.07	1.026	1.005352
26											0.909	1.11	1.01	0.995	1.006659
27											0.891	1.07	1.02	0.99	0.993851
28											0.915	1.23	0.98	1.004	1.032797
29											0.93	1.2	0.99	0.994	1.030243
30	5	2	7	12	6.5	82	78	95	117	93	0.921	1.23	0.98	0.979	1.028696
31											0.896	1.13	1	1.003	1.006965
32											0.909	1.13		1.005	1.015618
33											0.897	0.84		0.991	0.91081
34											0.848	0.81		1.027	0.895276
35	5	2	5	10	5.5	83	73	86	110	88	0.888	0.99	0.82	0.998	0.924325
36											0.907	0.89	0.9	0.999	0.922701
37											0.898	1.07	0.99	0.966	0.982663
38											0.867	1.14	1.01	0.964	0.996107
39											0.897	1.03	1	0.951	0.968958
40	6	2	7	10	6.25	88	75	95	107	91.25	0.885	1	1	0.988	0.969404
41											0.9	0.9	0.99	0.951	0.934913
42											0.888	0.71	1.01	0.938	0.886083
43											0.903	0.7	0.98	0.945	0.884088
44											0.887	0.82	0.97	0.932	0.902293
45	6	2	6	11	6.25	82	68	94	124	92	0.896	0.86	0.97	0.916	0.91273
46											0.882	0.89	0.92	0.949	0.910583
47											0.905	0.92	0.93	0.968	0.929968
48											0.892	0.93	0.94	0.96	0.929707
49											0.891	0.84	0.93	0.983	0.911582
50	6	2	5	15	7	83	68	86	125	90.5	0.888	0.8		0.999	0.896934
51											0.892	0.8		1.026	0.906548
52											0.901	0.79	0.86	1.025	0.893606
53											0.911	0.75	0.89	1.034	0.897605
54											0.878	0.75	0.96	1.058	0.911405
55	7	2	6	17	8	89	73	93	117	93	0.904	0.8	1	1.052	0.938974
56											0.904	0.94	0.99	1.079	0.978442
57											0.893	0.88	0.99	1.07	0.960392
58											0.913	0.77	0.98	1.082	0.93574
59											0.906	0.89	0.95	1.048	0.948884
60	7	2	6	16	7.75	84	67	93	121	91.25	0.907	0.81	0.91	1.031	0.915903

Appendix 16: Cardiopulmonary Responses to FES Cycling

Home Training Data

	VO2					VCO2					V					
timepoint	1	2	3	4	Average	1	2	3	4	Average	1	2	3	4	Average	% peak
Rest	0.294	0.163	0.27	0.29	0.255317	0.24	0.139	0.22	0.3	0.214744	8.4	5	6.6	9.44	7.340979	33.986
1	0.37	0.306	0.48	0.551	0.426309	0.3	0.276	0.4	0.5	0.363332	10	9.5	11	15.4	11.45977	53.0545
2	0.509	0.415	0.62	0.851	0.59862	0.41	0.447	0.61	1	0.618214	12	12	16	25.9	16.67405	77.1947
3	0.519	0.436	0.73	0.873	0.639606	0.45	0.602	0.92	1.2	0.793695	14	16	24	29.9	20.91832	96.8441
4	0.79	0.435	0.59	0.858	0.668019	0.74	0.662	0.8	1.2	0.842057	19	22	21	30.6	23.16875	107.263
5	0.834	0.482	0.63	0.825	0.692631	0.94	0.703	0.73	1.1	0.858428	24	26	20	28.7	24.58753	113.831
6	0.837	0.457	0.68	0.899	0.718611	0.94	0.681	0.83	1.1	0.885883	25	27	22	29.6	25.81586	119.518
7	0.839	0.486	0.6	0.903	0.70786	0.91	0.63	0.72	1.1	0.830007	24	24	18	29.8	23.96933	110.969
8	0.828	0.499	0.63	0.913	0.718815	0.86	0.646	0.73	1	0.818912	23	23	19	29.8	23.8455	110.396
9	0.812	0.558	0.59	0.919	0.720239	0.83	0.846	0.67	1	0.844861	23	30	17	29.8	25.04952	115.97
10	0.833	0.526	0.63	0.912	0.726435	0.87	0.743	0.71	1	0.828704	24	27	19	29.3	24.74445	114.558
11	0.801	0.606	0.66	0.931	0.749273	0.82	0.817	0.75	1	0.851031	23	29	20	29.3	25.51879	118.143
12	0.816	0.564	0.66	0.918	0.740155	0.82	0.708	0.73	1	0.820154	23	25	20	30	24.50401	113.445
13	0.807	0.566	0.65	0.92	0.736214	0.81	0.72	0.72	1	0.811346	23	26	20	29.6	24.46476	113.263
14	0.806	0.625	0.68	0.9	0.75252	0.79	0.771	0.76	1	0.832764	22	29	21	31.3	25.77639	119.335
15	0.8	0.552	0.66	0.915	0.732241	0.78	0.691	0.74	1	0.798601	22	27	20	30	24.69398	114.324
16	0.775	0.607	0.67	0.89	0.736453	0.75	0.855	0.73	1	0.826404	21	34	20	31.1	26.49776	122.675
17	0.798	0.583	0.67	0.924	0.743468	0.77	0.849	0.73	1	0.839036	22	39	21	30.6	28.04916	129.857
18	0.8	0.572	0.68	0.921	0.742849	0.76	0.851	0.73	1	0.833626	21	43	21	31.1	28.98926	134.21
19	0.769	0.557	0.68	0.9	0.725484	0.75	0.697	0.73	0.9	0.781769	22	34	21	29.5	26.41672	122.3
20	0.783	0.619	0.7	0.901	0.749533	0.75	0.688	0.75	1	0.788117	21	30	21	30.6	25.8224	119.548
21	0.778	0.605	0.68	0.872	0.733107	0.74	0.714	0.72	0.9	0.773293	21	32	20	29.7	25.77914	119.348
22	0.787	0.606	0.67	0.881	0.736201	0.74	0.735	0.71	0.9	0.776119	21	33	21	29.6	26.18801	121.241
23	0.757	0.553	0.67	0.881	0.715824	0.72	0.59	0.71	0.9	0.739191	20	27	21	29.9	24.44076	113.152
24	0.751	0.586	0.68	0.879	0.724123	0.71	0.643	0.72	0.9	0.743067	20	29	21	28.9	24.82332	114.923
25	0.723	0.555	0.67	0.898	0.710878	0.67	0.552	0.72	0.9	0.71514	20	26	21	29.6	24.02199	111.213
26	0.751	0.602	0.67	0.861	0.720999	0.68	0.667	0.68	0.9	0.720681	19	31	20	27.7	24.6259	114.009
27	0.75	0.539	0.67	0.906	0.715575	0.67	0.575	0.68	0.9	0.705544	19	26	20	28.7	23.51892	108.884
28	0.755	0.563	0.65	0.84	0.700553	0.69	0.688	0.64	0.8	0.714184	20	33	19	27.7	24.82937	114.951
29	0.752	0.545	0.63	0.853	0.695228	0.7	0.649	0.63	0.8	0.705348	20	31	18	27.9	24.29761	112.489
30	0.719	0.538	0.62	0.873	0.687063	0.66	0.662	0.61	0.9	0.696195	20	33	18	27.8	24.71604	114.426
31	0.707	0.53	0.66	0.864	0.690345	0.63	0.602	0.66	0.9	0.689041	19	31	19	28.2	24.49266	113.392
32	0.714	0.46		0.86	0.678056	0.65	0.529		0.9	0.680516	19	27	13	28	21.75767	100.73
33	0.72	0.4		0.886	0.668783	0.65	0.335		0.9	0.619448	19	18	15	28.1	20.03693	92.7636
34	0.69	0.409		0.868	0.655538	0.59	0.333		0.9	0.603004	17	15	17	29.1	19.56575	90.5822
35	0.728	0.298	0.59	0.873	0.621148	0.65	0.297	0.49	0.9	0.575715	19	15	18	28.3	19.9011	92.1347
36	0.719	0.521	0.57	0.849	0.6657	0.65	0.47	0.52	0.8	0.621555	19	20	18	27.3	21.24521	98.3575
37	0.703	0.525	0.58	0.872	0.670903	0.63	0.565	0.58	0.8	0.654538	19	28	18	27.2	22.94023	106.205
38	0.714	0.538	0.6	0.859	0.677421	0.62	0.614	0.6	0.8	0.666435	18	32	18	26.7	23.67656	109.614
39	0.722	0.483	0.63	0.842	0.670271	0.65	0.497	0.63	0.8	0.64433	19	26	18	26.1	22.25361	103.026
40	0.75	0.467	0.63	0.852	0.674188	0.66	0.466	0.63	0.8	0.649899	19	25	18	27.2	22.35054	103.475
41	0.739	0.467	0.64	0.855	0.674144	0.67	0.418	0.63	0.8	0.630893	19	20	17	26	20.65859	95.6416
42	0.74	0.403	0.61	0.84	0.649242	0.66	0.284	0.62	0.8	0.587305	19	12	17	25.4	18.54886	85.8744
43	0.736	0.432	0.59	0.839	0.649524	0.66	0.309	0.58	0.8	0.586906	19	12	17	26	18.72735	86.7007
44	0.708	0.5	0.6	0.841	0.663209	0.63	0.408	0.58	0.8	0.600693	19	17	18	25.6	19.71028	91.2513
45	0.706	0.534	0.6	0.83	0.666248	0.63	0.46	0.58	0.8	0.608372	18	19	18	24.7	19.86174	91.9525
46	0.701	0.44	0.64	0.836	0.654945	0.62	0.413	0.59	0.8	0.604179	18	15	18	25.9	19.13583	88.5918
47	0.757	0.592	0.64	0.832	0.70589	0.68	0.553	0.6	0.8	0.659815	20	25	14	26.3	21.38194	98.9905
48	0.738	0.394	0.65	0.846	0.656164	0.66	0.368	0.61	0.8	0.611735	19	18	17	25.8	19.98174	92.508
49	0.736	0.299	0.61	0.876	0.630357	0.66	0.256	0.57	0.9	0.584801	19	11	18	27.1	18.94467	87.7068
50	0.728	0.4		0.871	0.666376	0.65	0.323		0.9	0.613071	19	13	21	27.9	20.06328	92.8856
51	0.782	0.555		0.89	0.742484	0.7	0.447		0.9	0.685954	20	19	21	28.7	21.98141	101.766
52	0.801	0.471	0.57	0.905	0.686556	0.72	0.371	0.5	0.9	0.628794	20	15	21	29	21.1662	97.9917
53	0.792	0.4	0.83	0.928	0.737758	0.72	0.302	0.75	1	0.683767	20	12	19	29.7	20.2603	93.7977
54	0.799	0.426	0.63	0.941	0.699654	0.7	0.319	0.61	1	0.656395	20	12	19	30.2	20.0233	92.7005
55	0.816	0.366	0.69	0.948	0.706316	0.74	0.298	0.7	1	0.682563	21	12	17	30	19.9824	92.5111
56	0.806	0.503	0.71	0.954	0.74286	0.73	0.469	0.7	1	0.732893	21	18		31.7	23.35592	108.129
57	0.846	0.362	0.72	0.957	0.720829	0.76	0.321	0.71	1	0.703379	21	13		32	22.11851	102.401
58	0.804	0.32	0.68	0.95	0.688444	0.73	0.246	0.67	1	0.66869	21	9.1		32.7	20.7815	96.2106
59	0.793	0.425	0.68	0.969	0.715645	0.72	0.381	0.64	1	0.68865	20	16		32.6	22.89033	105.974
60	0.774	0.448	0.66	0.999	0.721151	0.7	0.378	0.6	1	0.678368	20	15		32.2	22.2025	102.789

Appendix 17: Motion Analysis

Upright Cycling

12 W				25 W				50 W			
RF	Bfem	Gmed	GastL	RF	Bfem	Gmed	GastL	RF	Bfem	Gmed	GastL
1.1671	0.214	0.2734	0.3357	0.8755	0.239	0.2598	0.2748	1.371	0.2594	0.291	0.3175
0.9817	0.199	0.301	0.2962	0.9296	0.2127	0.2729	0.3447	1.222	0.2581	0.279	0.3177
0.7726	0.236	0.2663	0.316	0.6474	0.2182	0.2339	0.2821	1.237	0.3092	0.274	0.3041
0.7512	0.205	0.2836	0.2698	0.5573	0.2022	0.2937	0.3001	1.124	0.3109	0.254	0.3292
0.8971	0.208	0.2793	0.3172	0.7235	0.2208	0.2615	0.2934	1.531	0.2796	0.264	0.2805
0.6933	0.202	0.2195	0.3171	0.9168	0.1871	0.2178	0.3332	1.5	0.2872	0.222	0.3071
0.6335	0.205	0.2517	0.3383	0.619	0.2294	0.2352	0.3658	1.323	0.2432	0.258	0.296
0.9599	0.215	0.2744	0.3236	0.6468	0.2306	0.299	0.3216	1.278	0.2657	0.241	0.2626
0.6473	0.221	0.2695	0.4179	0.6991	0.212	0.2857	0.2746	1.057	0.2702	0.265	0.3207
0.6268	0.235	0.2617	0.3967	0.5649	0.2225	0.2596	0.3454	1.294	0.3165	0.261	0.3233
0.5233	0.178	0.2445	0.5006	0.416	0.216	0.2593	0.3487	1.378	0.3035	0.236	0.349
0.6301	0.223	0.2505	0.4583	0.5221	0.2313	0.2525	0.4	0.917	0.3227	0.254	0.2998
0.4984	0.199	0.2402	0.4983	0.4598	0.2064	0.2431	0.5087	0.827	0.2465	0.281	0.387
0.5072	0.221	0.2772	0.5417	0.4551	0.2255	0.3287	0.4121	0.699	0.2564	0.288	0.491
0.4086	0.207	0.295	0.46	0.4249	0.2334	0.2571	0.4266	0.653	0.2622	0.305	0.3437
0.4052	0.221	0.3237	0.4514	0.3669	0.235	0.3299	0.4738	0.661	0.2914	0.31	0.315
0.4123	0.212	0.238	0.4343	0.3546	0.2243	0.2554	0.4983	0.805	0.2752	0.261	0.3607
0.3164	0.202	0.2817	0.4115	0.3277	0.2291	0.2926	0.5881	0.732	0.2555	0.28	0.4674
0.3333	0.219	0.3025	0.8217	0.3086	0.2255	0.2992	0.4434	0.655	0.3096	0.317	0.6004
0.3388	0.186	0.2485	0.5032	0.3633	0.2188	0.2521	0.4828	0.496	0.2672	0.299	0.431
0.3321	0.21	0.2755	0.589	0.3934	0.2521	0.2455	0.5532	0.717	0.2952	0.258	0.4701
0.3182	0.223	0.2572	0.5514	0.3082	0.2701	0.2496	0.5199	0.47	0.2809	0.268	0.431
0.3586	0.215	0.2528	0.6001	0.3094	0.272	0.2585	0.5204	0.476	0.3318	0.246	0.4542
0.344	0.236	0.2547	0.5181	0.3667	0.3365	0.2519	0.5387	0.446	0.2963	0.286	0.516
0.3226	0.239	0.2603	0.6879	0.4393	0.2936	0.2538	0.5275	0.399	0.32	0.286	0.5173
0.3336	0.24	0.2988	0.7663	0.3271	0.3093	0.2685	0.5445	0.504	0.3559	0.317	0.5922
0.3624	0.238	0.2861	0.7202	0.2967	0.349	0.2647	0.6296	0.42	0.4562	0.296	0.5461
0.3517	0.251	0.2625	0.8078	0.3823	0.4572	0.3156	0.687	0.395	0.4225	0.296	0.6566
0.3622	0.257	0.2632	0.6463	0.3126	0.31	0.2648	0.6492	0.396	0.5177	0.301	0.6252
0.3345	0.305	0.2537	0.6543	0.3154	0.4485	0.2377	0.8494	0.461	0.3766	0.272	0.6525
0.3475	0.314	0.279	0.9075	0.2679	0.487	0.2779	0.5666	0.395	0.5758	0.312	0.6503
0.291	0.386	0.2428	0.8387	0.2782	0.4981	0.2358	0.7448	0.349	0.5293	0.304	0.6461
0.3234	0.413	0.3031	0.991	0.3936	0.6323	0.3092	0.873	0.416	0.6936	0.314	0.8503
0.3233	0.418	0.3316	0.886	0.302	0.623	0.299	0.7498	0.433	0.6144	0.396	0.881
0.2751	0.512	0.2584	0.7066	0.325	0.5693	0.2598	0.911	0.393	0.4866	0.341	0.7602
0.3511	0.718	0.3016	0.8524	0.3673	0.7011	0.2931	0.9105	0.367	0.6706	0.307	0.7254
0.2447	0.685	0.246	1.0207	0.3017	0.7668	0.259	0.9987	0.402	0.5357	0.376	0.9928
0.2645	0.651	0.2711	0.9376	0.2471	0.8823	0.2319	1.0178	0.393	0.7562	0.277	0.8414
0.2385	0.722	0.2261	0.8855	0.3015	0.862	0.2792	0.9575	0.321	0.9513	0.346	0.8258
0.3508	0.827	0.2819	0.9532	0.2956	0.8823	0.242	1.1684	0.365	1.1377	0.316	1.0161
0.2617	0.644	0.2063	1.0362	0.2431	0.8446	0.272	1.159	0.313	0.8961	0.305	1.2939
0.2546	0.739	0.3005	1.1442	0.25	0.8312	0.2755	0.8678	0.293	0.9187	0.334	0.9439
0.2791	0.682	0.256	0.9863	0.3102	0.5997	0.2525	1.205	0.326	0.9159	0.302	0.835
0.2723	0.62	0.2781	0.9873	0.2508	0.6264	0.3005	0.8479	0.323	1.005	0.262	1.2324
0.3307	0.616	0.2912	0.8729	0.2797	0.8399	0.3068	1.0627	0.282	1.1343	0.284	1.0466
0.227	0.653	0.2892	0.8793	0.2757	0.6443	0.2768	0.818	0.265	0.8033	0.277	1.0637
0.2184	0.561	0.2487	0.7355	0.252	0.9689	0.2845	0.8901	0.266	0.7321	0.296	1.0533
0.2606	0.811	0.2471	1.0562	0.3523	0.653	0.2617	0.9485	0.277	0.8012	0.277	1.0074
0.2513	0.586	0.278	0.8891	0.2923	0.5511	0.2302	0.9516	0.255	0.6911	0.286	0.8914
0.2977	0.454	0.2104	1.075	0.308	0.5985	0.2796	0.8704	0.29	0.6613	0.238	0.949
0.2915	0.569	0.284	0.9667	0.2942	0.6177	0.2764	1.1441	0.283	0.758	0.315	0.9059
0.2712	0.392	0.2431	0.9438	0.238	0.5568	0.2563	1.0674	0.236	0.6843	0.257	1.0776

Appendix 17: Motion Analysis

Upright Cycling

0.2709	0.525	0.2669	0.8741	0.2529	0.5588	0.2771	0.8866	0.239	0.6509	0.291	1.2865
0.2747	0.412	0.2667	0.9148	0.284	0.5109	0.2687	0.7995	0.274	0.6763	0.291	0.8569
0.2972	0.392	0.2529	0.956	0.2896	0.5444	0.2616	1.1776	0.274	0.5894	0.264	1.1391
0.2647	0.342	0.2598	1.0957	0.278	0.5767	0.2875	1.2041	0.317	0.5516	0.313	1.1908
0.2681	0.344	0.2753	0.9779	0.2581	0.3973	0.259	1.0763	0.26	0.68	0.272	0.9455
0.2561	0.4	0.2881	1.2885	0.2395	0.3975	0.2357	0.9724	0.316	0.6349	0.305	1.1627
0.2262	0.381	0.2446	0.8789	0.262	0.3548	0.2579	1.0689	0.223	0.5338	0.276	1.0111
0.2835	0.332	0.2497	1.2211	0.2656	0.4253	0.2683	1.0438	0.238	0.5014	0.285	1.0632
0.3003	0.377	0.2448	0.979	0.3271	0.3106	0.1998	1.045	0.27	0.4442	0.282	0.927
0.2871	0.298	0.2831	0.8577	0.2847	0.3254	0.2704	1.1198	0.29	0.5512	0.278	0.7448
0.2371	0.341	0.3105	0.9015	0.3382	0.3163	0.2802	1.5716	0.276	0.431	0.329	1.0422
0.2316	0.259	0.2838	1.0546	0.2918	0.2583	0.2468	0.956	0.301	0.4303	0.288	1.4417
0.2761	0.27	0.2709	1.1872	0.361	0.2803	0.3059	1.0782	0.282	0.4755	0.273	1.1835
0.2261	0.231	0.2433	1.1568	0.2666	0.2912	0.271	0.8358	0.276	0.4602	0.288	1.2142
0.2429	0.222	0.2719	0.8475	0.2744	0.248	0.2827	0.9398	0.314	0.4575	0.263	0.8294
0.292	0.235	0.2871	1.0294	0.2987	0.2568	0.2725	1.1615	0.248	0.348	0.307	1.001
0.3007	0.227	0.2617	0.9334	0.33	0.2579	0.2639	1.0938	0.234	0.3601	0.29	1.1683
0.3057	0.228	0.2756	1.0139	0.3474	0.2323	0.3004	1.0234	0.27	0.4023	0.281	0.9712
0.335	0.293	0.3158	0.9197	0.3038	0.2403	0.2815	0.7133	0.324	0.3365	0.271	0.8326
0.3706	0.246	0.2898	0.8779	0.3175	0.232	0.2666	0.9661	0.308	0.3809	0.266	1.0416
0.3619	0.217	0.2917	0.7928	0.469	0.2564	0.2368	1.2362	0.35	0.3193	0.288	1.1836
0.3938	0.249	0.2781	0.8789	0.3967	0.3021	0.2491	0.7324	0.361	0.3436	0.307	1.0687
0.5213	0.241	0.2752	0.7675	0.4276	0.2598	0.251	0.635	0.359	0.3645	0.25	0.9394
0.4371	0.236	0.2712	0.8523	0.487	0.2668	0.3052	0.655	0.4	0.364	0.296	0.7749
0.5082	0.241	0.2603	0.615	0.5327	0.2319	0.2524	0.7642	0.655	0.3873	0.273	0.8791
0.6781	0.236	0.2765	0.7818	0.4831	0.2399	0.2849	0.5167	0.542	0.2704	0.262	0.8013
0.6855	0.216	0.2578	0.4778	0.5323	0.3299	0.2693	0.4648	0.659	0.2485	0.283	1.0032
0.6893	0.242	0.2967	0.4092	0.6107	0.2418	0.2834	0.5446	0.902	0.3189	0.316	0.7266
0.6139	0.226	0.2478	0.578	0.6112	0.2218	0.2954	0.4999	0.864	0.2729	0.296	0.7094
0.5934	0.241	0.3278	0.5492	0.5709	0.2889	0.3217	0.4985	0.795	0.2628	0.27	0.5374
0.7873	0.245	0.2595	0.4008	0.6218	0.2936	0.2506	0.4804	0.752	0.3483	0.306	0.5287
0.8196	0.282	0.339	0.42	0.7584	0.3126	0.2925	0.4291	0.64	0.3084	0.253	0.4735
1.0286	0.236	0.2437	0.3915	0.9457	0.2951	0.2553	0.3473	1.031	0.2156	0.259	0.3173
0.8393	0.315	0.2597	0.325	0.8794	0.315	0.284	0.3786	1.086	0.229	0.278	0.3889
0.9126	0.267	0.2568	0.3473	0.9662	0.2875	0.2369	0.3475	0.964	0.2785	0.314	0.3778
1.0567	0.266	0.2451	0.3348	0.8348	0.3408	0.3055	0.3449	1.078	0.2482	0.275	0.3237
0.9721	0.238	0.2701	0.3642	1.1531	0.3241	0.2335	0.3341	1.032	0.3101	0.285	0.3802
0.8634	0.247	0.2672	0.3451	1.2031	0.2597	0.2731	0.3357	1.026	0.3073	0.282	0.3473
1.4313	0.276	0.2936	0.3386	1.0001	0.313	0.3007	0.3348	1.531	0.2983	0.286	0.3008
0.957	0.224	0.2845	0.2859	1.1763	0.2977	0.255	0.3582	1.269	0.2824	0.284	0.3039
1.0925	0.241	0.2544	0.2889	1.0901	0.2922	0.2581	0.3268	1.439	0.3007	0.239	0.3416
1.0773	0.242	0.3109	0.3442	1.0244	0.2777	0.2487	0.3108	1.399	0.2746	0.257	0.3007
1.407	0.231	0.2671	0.3039	1.2049	0.2637	0.2684	0.2917	1.483	0.28	0.254	0.3025
1.2385	0.232	0.2589	0.3109	1.107	0.2318	0.2469	0.3151	1.216	0.2169	0.282	0.2724
1.3639	0.213	0.2514	0.2637	1.1641	0.2603	0.2695	0.3517	1.531	0.229	0.298	0.2891
0.9818	0.248	0.2675	0.3348	1.3983	0.2489	0.2849	0.3427	1.413	0.2094	0.327	0.3202
1.353	0.237	0.238	0.2975	1.065	0.2192	0.2921	0.3881	1.45	0.2116	0.288	0.309
1.0339	0.25	0.3405	0.3182	1.0301	0.2273	0.2515	0.2556	1.623	0.2906	0.264	0.3368
1.0205	0.232	0.2509	0.3367	0.8804	0.2052	0.2821	0.3359	1.55	0.2596	0.283	0.28

Appendix 17: Motion Analysis

Upright Cycling

75 W				100 W			
RF	Bfem	Gmed	GastL	RF	Bfem	Gmed	GastL
1.498	0.2854	0.245	0.3283	1.881	0.3268	0.3134	0.318
1.478	0.3089	0.282	0.3479	1.999	0.3429	0.2597	0.303
1.872	0.2644	0.281	0.3133	2.241	0.3407	0.2453	0.382
1.912	0.3195	0.267	0.2858	2.616	0.2707	0.2931	0.319
1.85	0.2918	0.334	0.3117	2.075	0.296	0.3338	0.376
1.675	0.2502	0.265	0.3269	1.784	0.3068	0.2659	0.349
1.777	0.2743	0.297	0.3332	2.132	0.3422	0.3407	0.319
2.1	0.3157	0.242	0.2859	2.278	0.3266	0.291	0.356
2.055	0.296	0.291	0.3548	2.143	0.3521	0.2672	0.382
1.719	0.3825	0.34	0.3232	2.21	0.3029	0.3293	0.343
1.551	0.3415	0.271	0.3507	2.199	0.3823	0.3327	0.359
1.081	0.3043	0.306	0.3683	1.991	0.3736	0.3621	0.47
0.846	0.2536	0.294	0.459	2.07	0.3813	0.3334	0.491
0.979	0.3112	0.303	0.3685	1.708	0.3773	0.3826	0.508
0.887	0.2495	0.318	0.3914	1.773	0.346	0.3433	0.434
0.855	0.2567	0.312	0.4023	1.54	0.3147	0.3977	0.496
0.914	0.3293	0.341	0.5558	1.04	0.4261	0.3681	0.54
0.768	0.2891	0.355	0.6547	1.697	0.3172	0.3838	0.707
0.729	0.2915	0.318	0.4322	1.339	0.3988	0.3694	0.482
0.758	0.3324	0.316	0.5074	0.993	0.4131	0.3596	0.549
0.59	0.3777	0.262	0.5003	0.852	0.37	0.4259	0.527
0.612	0.3735	0.31	0.4383	0.67	0.4933	0.3864	0.515
0.564	0.3417	0.332	0.493	0.749	0.3698	0.3752	0.57
0.541	0.3875	0.337	0.4987	0.733	0.4277	0.3804	0.514
0.569	0.4128	0.353	0.6048	0.747	0.4229	0.4241	0.594
0.513	0.5266	0.312	0.5715	0.685	0.6019	0.492	0.658
0.519	0.4392	0.325	0.5728	0.699	0.4415	0.4611	0.869
0.47	0.5524	0.314	0.6907	0.766	0.6611	0.4573	0.738
0.498	0.5379	0.317	0.7711	0.605	0.4573	0.5051	0.647
0.517	0.6937	0.337	0.8712	0.612	0.6971	0.4266	0.725
0.501	0.5588	0.343	0.8065	0.615	0.5458	0.4162	0.657
0.513	0.5753	0.361	0.9239	0.579	0.6432	0.3484	0.852
0.547	0.6471	0.38	0.9576	0.626	0.6756	0.4092	0.755
0.575	0.7926	0.373	0.787	0.487	0.6457	0.4769	0.948
0.551	0.8293	0.399	0.8212	0.567	0.5841	0.4541	0.979
0.449	0.7776	0.346	0.8536	0.566	0.6386	0.4075	0.915
0.493	0.9397	0.301	0.8081	0.446	0.7756	0.3948	0.753
0.382	0.9037	0.311	0.8757	0.421	0.7882	0.3826	1.07
0.43	0.8246	0.288	1.0088	0.457	0.7967	0.3624	1.295
0.353	0.8505	0.312	1.0339	0.319	0.7579	0.311	1.039
0.315	1.2319	0.282	0.9287	0.381	1.0484	0.2968	1.075
0.3	1.0298	0.283	1.2948	0.337	0.7381	0.2951	0.914
0.345	0.9311	0.291	1.2067	0.398	0.7601	0.3218	1.034
0.314	1.155	0.303	0.9233	0.343	0.9766	0.3293	0.678
0.314	0.9169	0.251	1.0347	0.315	0.6855	0.287	0.832
0.295	1.043	0.305	0.8273	0.306	0.9261	0.2873	0.779
0.283	0.8942	0.277	0.7468	0.263	1.1508	0.2824	0.786
0.253	1.0547	0.273	1.1297	0.342	1.1095	0.3243	1.036
0.298	0.9465	0.26	1.0853	0.336	0.8929	0.3199	0.941
0.286	0.8878	0.281	0.9904	0.24	0.8968	0.3086	0.942
0.286	0.7906	0.31	1.0384	0.281	0.8748	0.3238	1.065
0.275	0.9501	0.312	0.9738	0.266	1.1029	0.2966	1.066

Appendix 17: Motion Analysis

Upright Cycling

0.302	0.7646	0.262	1.0536	0.267	1.0001	0.2951	0.974
0.271	0.771	0.308	1.4337	0.236	1.0289	0.2956	1.025
0.309	0.6731	0.29	1.0657	0.332	0.7401	0.3137	1.133
0.281	0.8661	0.324	1.25	0.28	0.8712	0.3175	1.204
0.229	0.8968	0.272	1.2519	0.32	0.8682	0.274	1.415
0.274	0.8736	0.284	1.2858	0.257	0.8169	0.309	1.108
0.245	0.7402	0.315	1.1969	0.328	0.8508	0.2901	1.224
0.261	0.6475	0.271	1.3171	0.265	0.6035	0.2978	1.244
0.282	0.6232	0.254	0.9576	0.287	0.6807	0.2905	0.952
0.268	0.4863	0.267	1.0604	0.243	0.6555	0.3039	0.902
0.28	0.4595	0.286	1.579	0.343	0.5211	0.2905	1.656
0.291	0.4876	0.275	0.9987	0.294	0.5508	0.284	1.127
0.317	0.4833	0.332	1.2119	0.302	0.6534	0.3257	1.267
0.312	0.482	0.326	0.9667	0.327	0.5879	0.3095	1.125
0.293	0.3699	0.25	1.0937	0.289	0.4993	0.2985	1.068
0.322	0.3927	0.286	0.9692	0.315	0.65	0.3156	0.919
0.346	0.4379	0.262	1.311	0.357	0.5509	0.279	1.101
0.4	0.3312	0.287	1.1171	0.347	0.5147	0.3292	1.106
0.425	0.4492	0.318	0.7606	0.403	0.4742	0.3277	0.95
0.351	0.4335	0.28	1.1915	0.356	0.4282	0.2898	1.113
0.475	0.3762	0.261	1.3931	0.58	0.4236	0.3302	1.339
0.381	0.3992	0.253	0.9889	0.497	0.4224	0.3095	0.926
0.458	0.3414	0.308	0.77	0.542	0.4611	0.3073	0.855
0.519	0.3997	0.255	0.6661	0.528	0.4494	0.3438	0.829
0.593	0.3374	0.265	0.8505	0.434	0.3896	0.2882	0.909
0.686	0.2932	0.271	0.7232	0.613	0.3935	0.2948	0.877
1.027	0.3424	0.303	0.7189	0.623	0.3623	0.3307	0.859
0.646	0.2675	0.28	0.5296	0.618	0.3641	0.2761	0.706
0.626	0.3205	0.263	0.4351	0.657	0.2995	0.3133	0.6
0.663	0.3383	0.343	0.5614	0.664	0.396	0.3408	0.546
0.687	0.3146	0.258	0.4171	0.737	0.3209	0.2863	0.465
1.006	0.3581	0.321	0.3871	0.891	0.3547	0.3489	0.375
1.025	0.3589	0.307	0.3656	0.921	0.298	0.3115	0.439
1.151	0.338	0.32	0.3285	1.007	0.3885	0.3972	0.379
1.155	0.3243	0.282	0.3732	1.047	0.2471	0.2722	0.366
0.935	0.3532	0.319	0.3161	1.204	0.2501	0.3032	0.405
1.069	0.3881	0.295	0.2793	1.071	0.2712	0.3514	0.393
1.174	0.3665	0.283	0.33	1.247	0.274	0.3342	0.36
1.403	0.3049	0.284	0.3322	1.142	0.2638	0.3182	0.373
1.351	0.3464	0.27	0.3451	1.157	0.3137	0.3223	0.372
1.649	0.3038	0.26	0.3337	1.481	0.28	0.3062	0.353
1.556	0.2808	0.259	0.2884	1.379	0.31	0.2778	0.31
1.426	0.2397	0.242	0.3372	1.731	0.2637	0.2983	0.277
2.13	0.2477	0.275	0.3505	1.819	0.288	0.2904	0.391
1.809	0.2345	0.285	0.3186	2.071	0.3	0.297	0.39
1.938	0.2277	0.291	0.3065	2.261	0.2447	0.3306	0.31
2.042	0.2663	0.258	0.3229	2.009	0.2825	0.3149	0.361
1.671	0.2684	0.329	0.331	2.022	0.2705	0.3012	0.252
1.684	0.2337	0.286	0.3714	2.49	0.2717	0.3132	0.367

Appendix 17: Motion Analysis

Recumbent Cycling

12 W				25 W				50 W			
RF	Bfem	Gmed	GastL	RF	Bfem	Gmed	GastL	RF	Bfem	Gmed	GastL
0.4147	0.261	0.4266	0.406	0.489	0.4153	0.386	0.3807	0.869	0.3228	0.453	0.5463
0.492	0.283	0.5395	0.3964	0.5762	0.3967	0.3803	0.4567	0.957	0.4108	0.513	0.5244
0.4816	0.266	0.4918	0.4369	0.5342	0.4167	0.4039	0.4108	0.761	0.4311	0.461	0.4944
0.5363	0.268	0.5799	0.3803	0.5887	0.4918	0.4549	0.406	0.858	0.4526	0.562	0.4803
0.473	0.247	0.4827	0.3978	0.6097	0.3916	0.374	0.4829	0.91	0.4465	0.512	0.4711
0.4023	0.293	0.5475	0.388	0.6026	0.4436	0.4117	0.3989	0.807	0.4724	0.533	0.4649
0.451	0.248	0.5175	0.4102	0.6033	0.4264	0.3562	0.3825	0.819	0.4697	0.477	0.5012
0.3962	0.261	0.5252	0.3737	0.532	0.3886	0.386	0.4435	0.798	0.438	0.506	0.3952
0.4141	0.38	0.4815	0.4416	0.6029	0.4183	0.4423	0.439	0.868	0.4926	0.524	0.4789
0.4123	0.298	0.5036	0.3731	0.5747	0.3855	0.436	0.4176	0.85	0.4885	0.478	0.4409
0.3292	0.297	0.5356	0.4011	0.582	0.3948	0.4311	0.4477	0.813	0.4053	0.517	0.4051
0.4291	0.345	0.4348	0.4201	0.5493	0.3711	0.402	0.4397	0.846	0.4606	0.521	0.4547
0.4301	0.335	0.608	0.4081	0.5718	0.4058	0.3314	0.4315	0.776	0.4445	0.495	0.4685
0.4152	0.346	0.4016	0.4716	0.5141	0.4144	0.342	0.3733	0.79	0.4337	0.536	0.417
0.4208	0.341	0.612	0.4016	0.5242	0.4585	0.3865	0.4028	0.758	0.4284	0.594	0.4141
0.3997	0.345	0.4765	0.4241	0.4932	0.4093	0.4513	0.4542	0.87	0.4544	0.635	0.4422
0.3414	0.338	0.5892	0.3925	0.4845	0.4518	0.4961	0.3862	0.747	0.4182	0.507	0.4307
0.3598	0.308	0.5086	0.4194	0.5961	0.4929	0.438	0.4594	0.622	0.5101	0.537	0.4654
0.3368	0.319	0.4941	0.4177	0.4873	0.4614	0.3683	0.4115	0.681	0.4749	0.579	0.4635
0.3619	0.273	0.4933	0.3948	0.5177	0.4476	0.3688	0.4582	0.694	0.4871	0.579	0.466
0.3239	0.321	0.4903	0.4519	0.4574	0.4394	0.4203	0.3831	0.686	0.4515	0.57	0.4259
0.3062	0.303	0.5605	0.4208	0.4686	0.4148	0.4058	0.4571	0.778	0.5251	0.583	0.5155
0.2813	0.311	0.4385	0.3915	0.4699	0.4478	0.4265	0.4039	0.7	0.5561	0.612	0.5394
0.3328	0.306	0.5838	0.3867	0.5586	0.4734	0.4199	0.4041	0.701	0.5692	0.534	0.4735
0.3607	0.311	0.4816	0.4631	0.5192	0.4819	0.4551	0.4422	0.667	0.552	0.486	0.5553
0.3591	0.284	0.5784	0.3885	0.5544	0.4338	0.4222	0.5139	0.65	0.4948	0.526	0.5364
0.3366	0.35	0.5097	0.4218	0.5044	0.5115	0.4294	0.4319	0.699	0.7266	0.561	0.4542
0.3129	0.312	0.4609	0.5026	0.4302	0.4835	0.3644	0.3917	0.727	0.4955	0.591	0.4834
0.259	0.297	0.5219	0.3925	0.4642	0.4623	0.444	0.4235	0.626	0.574	0.604	0.5136
0.3152	0.302	0.4247	0.4499	0.4418	0.4411	0.4213	0.4266	0.591	0.5313	0.56	0.5723
0.2359	0.346	0.5642	0.4613	0.3943	0.4966	0.3846	0.4857	0.611	0.6053	0.506	0.5292
0.31	0.338	0.4596	0.4646	0.5308	0.4261	0.4311	0.5436	0.597	0.6234	0.57	0.6011
0.3325	0.312	0.5695	0.5529	0.5429	0.5572	0.3936	0.5454	0.641	0.6237	0.532	0.7431
0.3011	0.361	0.4931	0.4445	0.4929	0.5957	0.3797	0.6167	0.721	0.6162	0.536	0.7673
0.2878	0.438	0.5501	0.5588	0.443	0.5196	0.4415	0.4825	0.568	0.7372	0.54	0.674
0.3652	0.405	0.5532	0.5543	0.4009	0.6059	0.4408	0.5589	0.551	0.7135	0.544	0.6799
0.3703	0.375	0.4619	0.522	0.3107	0.6382	0.407	0.5573	0.548	0.7061	0.539	0.6053
0.27	0.443	0.4957	0.569	0.5073	0.7312	0.4068	0.6556	0.59	0.7614	0.481	0.6131
0.324	0.513	0.4226	0.5274	0.5843	0.6485	0.4082	0.6542	0.67	0.7896	0.536	0.7118
0.3132	0.621	0.5543	0.4783	0.424	0.7904	0.3888	0.5466	0.48	0.7756	0.589	0.7033
0.2546	0.525	0.4473	0.5894	0.4368	0.7387	0.405	0.5795	0.5	1.0887	0.564	0.8537
0.2913	0.602	0.562	0.5396	0.3868	0.7866	0.3753	0.6201	0.533	0.9525	0.516	0.8025
0.2843	0.515	0.4258	0.51	0.4856	0.7501	0.3551	0.6622	0.599	0.918	0.505	0.7937
0.3317	0.582	0.5399	0.646	0.4002	0.8411	0.3952	0.6722	0.52	0.9888	0.56	0.6929
0.3129	0.613	0.4562	0.6471	0.4801	0.8548	0.4017	0.667	0.571	1.0834	0.509	0.9165
0.3066	0.616	0.5504	0.6077	0.4612	0.7333	0.3696	0.6175	0.579	0.8643	0.496	0.8409
0.4701	0.504	0.4349	0.515	0.3959	0.6542	0.3859	0.7837	0.486	1.028	0.511	1.0645
0.3672	0.571	0.5811	0.7018	0.4321	0.8116	0.3892	0.7031	0.532	1.0474	0.513	0.8972
0.3812	0.673	0.4501	0.4831	0.4129	0.7437	0.4705	0.5996	0.332	0.9421	0.482	0.777
0.4026	0.55	0.4971	0.6505	0.5074	0.9237	0.4913	0.781	0.334	1.4204	0.464	0.8557
0.3813	0.763	0.5238	0.6448	0.5002	0.8896	0.4291	0.7542	0.356	1.1077	0.543	0.8415
0.3125	0.772	0.4264	0.6572	0.4378	0.8658	0.3844	0.6769	0.556	0.9845	0.466	0.8966

Appendix 17: Motion Analysis

Recumbent Cycling

0.3149	0.714	0.5161	0.7444	0.4521	0.8193	0.3546	0.7107	0.552	1.0829	0.523	1.0766
0.3532	0.701	0.554	0.8882	0.4811	0.75	0.4317	0.8829	0.565	1.1701	0.557	0.9091
0.3443	0.757	0.4759	0.7705	0.5145	0.6639	0.4138	0.8315	0.557	1.0057	0.582	0.8826
0.2921	0.525	0.4902	0.6489	0.4292	0.8033	0.3766	0.7402	0.471	0.9593	0.437	0.9118
0.3187	0.585	0.5551	0.6423	0.4334	0.7677	0.4311	0.7629	0.553	1.0122	0.438	0.8749
0.2609	0.46	0.4801	0.7725	0.4495	0.7177	0.3999	0.7594	0.543	0.8233	0.467	0.8732
0.27	0.474	0.5388	0.5326	0.3747	0.6104	0.435	0.7169	0.51	1.0089	0.524	0.8825
0.3316	0.585	0.4399	0.6468	0.3734	0.7459	0.3596	0.858	0.575	0.9939	0.58	0.843
0.275	0.466	0.589	0.5939	0.4469	0.7281	0.3804	0.6373	0.553	0.9558	0.526	0.7838
0.3295	0.449	0.4346	0.6528	0.4437	0.724	0.3401	0.7761	0.523	0.7651	0.445	0.804
0.3167	0.453	0.5633	0.715	0.4815	0.689	0.4148	0.8061	0.531	0.6558	0.435	0.8217
0.3459	0.506	0.3838	0.7428	0.4231	0.6233	0.4334	0.8107	0.521	0.6796	0.456	0.8617
0.3336	0.446	0.5379	0.5192	0.3966	0.6438	0.367	0.7119	0.552	0.7519	0.51	0.9867
0.288	0.324	0.4082	0.5308	0.3895	0.5536	0.3514	0.7319	0.511	0.7793	0.517	0.8477
0.3404	0.347	0.5747	0.5287	0.4126	0.5241	0.3821	0.7785	0.539	0.615	0.498	0.6797
0.3047	0.322	0.4804	0.5749	0.431	0.5032	0.407	0.7609	0.559	0.5389	0.446	0.7242
0.3117	0.275	0.4653	0.5858	0.425	0.58	0.4228	0.6763	0.511	0.5909	0.448	0.6764
0.3428	0.226	0.492	0.5198	0.4487	0.4688	0.406	0.606	0.557	0.647	0.457	0.8316
0.3211	0.292	0.516	0.4577	0.489	0.5342	0.3597	0.5884	0.587	0.5944	0.524	0.5986
0.3488	0.278	0.4948	0.5636	0.4536	0.489	0.3942	0.6808	0.595	0.4911	0.575	0.813
0.3691	0.308	0.4516	0.4745	0.4755	0.3821	0.3557	0.55	0.497	0.5045	0.494	0.6326
0.2918	0.332	0.4837	0.4105	0.4103	0.4759	0.416	0.5662	0.511	0.4079	0.468	0.6229
0.3233	0.291	0.4342	0.5441	0.4824	0.3974	0.4343	0.5434	0.571	0.4932	0.446	0.6211
0.3778	0.27	0.5095	0.3943	0.4049	0.4414	0.4101	0.5735	0.505	0.5028	0.491	0.5761
0.3253	0.237	0.5786	0.4606	0.4878	0.3678	0.3585	0.5237	0.587	0.4214	0.5	0.6068
0.2957	0.282	0.4373	0.3934	0.4368	0.396	0.376	0.4328	0.561	0.4028	0.537	0.6205
0.3547	0.263	0.619	0.4751	0.4709	0.402	0.4115	0.4876	0.585	0.4066	0.494	0.5381
0.3248	0.272	0.4388	0.4832	0.4952	0.4027	0.4316	0.4757	0.647	0.3879	0.429	0.579
0.3609	0.32	0.6385	0.4069	0.5245	0.3507	0.419	0.4512	0.628	0.3791	0.433	0.5794
0.2456	0.305	0.4276	0.4327	0.4846	0.356	0.3914	0.454	0.587	0.4042	0.481	0.5668
0.3167	0.319	0.5992	0.4117	0.5143	0.3482	0.4214	0.4414	0.679	0.3747	0.525	0.624
0.3487	0.28	0.4184	0.4241	0.5237	0.3674	0.4205	0.4063	0.571	0.3197	0.553	0.582
0.3379	0.299	0.5857	0.407	0.5146	0.3693	0.3431	0.4593	0.624	0.3471	0.488	0.4699
0.3559	0.248	0.478	0.4484	0.5826	0.3821	0.3877	0.453	0.702	0.3997	0.487	0.4402
0.386	0.286	0.5372	0.4032	0.635	0.3622	0.4138	0.4231	0.696	0.4192	0.476	0.4129
0.4629	0.273	0.5164	0.4294	0.6802	0.3289	0.4178	0.4173	0.866	0.4752	0.484	0.5464
0.4294	0.284	0.5614	0.3563	0.5783	0.3287	0.3401	0.4023	0.868	0.4518	0.547	0.5633
0.426	0.272	0.5298	0.4255	0.6488	0.3358	0.3446	0.3901	0.871	0.4777	0.538	0.4323
0.4846	0.279	0.5146	0.4175	0.7481	0.3668	0.3609	0.4297	0.89	0.3932	0.536	0.3822
0.4257	0.289	0.5253	0.4164	0.6798	0.3891	0.4112	0.4592	0.805	0.4554	0.498	0.4119
0.5131	0.292	0.4883	0.3981	0.6077	0.3723	0.3948	0.4194	0.932	0.4456	0.472	0.485
0.443	0.284	0.5076	0.4456	0.6175	0.3983	0.4829	0.3795	0.965	0.4594	0.555	0.5059
0.3817	0.254	0.4493	0.4261	0.6689	0.3961	0.4295	0.4002	0.859	0.3444	0.524	0.5219
0.4112	0.271	0.5226	0.4306	0.677	0.4018	0.3724	0.4231	0.792	0.4173	0.499	0.4255
0.5199	0.297	0.5754	0.4335	0.6276	0.3938	0.3835	0.4184	0.794	0.4174	0.517	0.4762
0.4563	0.285	0.556	0.4682	0.5859	0.3691	0.368	0.3954	0.844	0.4537	0.484	0.4321
0.4672	0.255	0.4737	0.4145	0.6304	0.3808	0.4306	0.4343	0.833	0.4334	0.503	0.4505
0.4551	0.228	0.5317	0.387	0.4969	0.4662	0.4258	0.4086	0.96	0.3899	0.558	0.6231
0.5087	0.242	0.5139	0.3784	0.6429	0.4426	0.4058	0.4276	0.864	0.3789	0.481	0.5905

Appendix 17: Motion Analysis

Recumbent Cycling

75 W				100 W			
RF	Bfem	Gmed	GastL	RF	Bfem	Gmed	GastL
1.351	0.4029	0.547	0.4746	0.984	0.4181	0.5041	0.463
1.532	0.3675	0.586	0.4739	1.436	0.3937	0.4732	0.579
1.52	0.4727	0.574	0.496	1.403	0.4942	0.5307	0.545
1.192	0.3987	0.51	0.4673	1.471	0.4648	0.5856	0.54
1.216	0.4273	0.484	0.4506	1.371	0.49	0.544	0.535
1.322	0.4324	0.525	0.498	1.64	0.4728	0.5299	0.568
1.322	0.5075	0.617	0.4801	1.468	0.5084	0.5751	0.621
1.478	0.5111	0.645	0.4869	1.739	0.4743	0.5695	0.536
1.414	0.4384	0.56	0.5104	1.536	0.5771	0.6391	0.461
1.363	0.4121	0.532	0.4719	1.435	0.5404	0.5888	0.514
1.556	0.46	0.609	0.5349	1.551	0.6025	0.6341	0.454
1.339	0.3683	0.635	0.5054	1.405	0.5256	0.6614	0.517
1.488	0.5402	0.796	0.4806	1.811	0.5567	0.7497	0.553
1.397	0.5603	0.731	0.5036	1.324	0.5759	0.7091	0.626
1.015	0.597	0.791	0.5918	1.573	0.5379	0.6636	0.601
0.898	0.6784	0.703	0.5727	1.304	0.6157	0.7139	0.588
1.196	0.5834	0.629	0.5106	1.482	0.5463	0.6648	0.485
1.074	0.6265	0.743	0.4867	1.272	0.6451	0.7959	0.564
1.08	0.5834	0.835	0.5604	1.329	0.7296	0.8564	0.679
1.131	0.5544	0.699	0.4859	0.89	0.7531	0.6849	0.579
1.1	0.5446	0.738	0.5132	0.858	0.87	0.7909	0.524
0.878	0.7112	0.693	0.5197	0.906	0.7176	0.8876	0.429
0.94	0.7646	0.747	0.5108	0.838	0.7437	0.7328	0.552
0.84	0.7634	0.847	0.4957	0.811	0.6941	0.9005	0.571
0.871	0.8626	0.783	0.5595	0.818	0.7748	0.9356	0.637
0.756	0.809	0.686	0.6099	0.855	0.854	0.9375	0.623
0.865	0.8601	0.667	0.5199	0.636	1.016	0.8986	0.622
0.7	0.7543	0.709	0.6174	0.798	0.8032	0.7168	0.491
0.715	0.847	0.657	0.5602	0.639	0.7414	0.7057	0.541
0.742	0.8472	0.752	0.577	0.76	0.906	0.9535	0.618
0.803	0.8837	0.709	0.5804	0.607	1.0185	1.0324	0.52
0.724	0.959	0.646	0.6888	0.54	1.0777	0.7855	0.654
0.565	0.8181	0.631	0.8768	0.681	1.0754	0.729	0.774
0.65	0.9707	0.696	0.7288	0.549	0.9754	0.7431	0.728
0.634	1.1204	0.693	0.8024	0.549	0.874	0.7513	0.737
0.6	0.9615	0.746	0.8497	0.509	1.1056	0.8667	0.67
0.591	0.9884	0.629	0.8399	0.64	0.9628	0.8536	0.657
0.362	1.0301	0.583	0.8312	0.612	1.0088	0.8032	0.984
0.308	1.1583	0.614	0.8827	0.559	1.2498	0.7727	0.891
0.321	1.1622	0.607	0.7433	0.502	0.97	0.6052	0.881
0.306	0.9437	0.732	0.7758	0.515	1.1973	0.7374	0.855
0.281	1.0046	0.648	0.9425	0.426	1.2222	0.6289	0.968
0.25	1.1308	0.607	0.8375	0.468	0.9753	0.6679	0.922
0.271	1.0826	0.581	0.9545	0.444	1.1613	0.681	0.834
0.286	1.2362	0.529	0.9573	0.384	0.9856	0.6349	0.896
0.263	1.1857	0.445	0.8665	0.41	1.2682	0.4966	0.984
0.214	1.182	0.526	0.8737	0.343	1.287	0.4758	0.973
0.268	1.21	0.566	0.9996	0.462	1.0746	0.542	0.826
0.194	1.0605	0.585	0.8765	0.452	1.0437	0.5395	0.702
0.24	0.9814	0.482	0.9505	0.37	1.2758	0.5317	0.764
0.26	1.1353	0.417	0.9299	0.454	1.1637	0.5417	0.959
0.267	1.0792	0.503	1.3129	0.474	1.1584	0.4896	1.059

Appendix 17: Motion Analysis

Recumbent Cycling

0.284	1.0703	0.553	1.2834	0.295	1.2629	0.5113	0.946
0.296	0.9963	0.561	1.3126	0.318	1.205	0.5559	1.2
0.247	1.1659	0.563	1.1661	0.311	1.3028	0.5817	1.168
0.481	1.1728	0.475	0.9087	0.325	1.3125	0.5128	0.997
0.594	1.0849	0.505	0.9424	0.512	1.2302	0.5348	1.033
0.435	0.846	0.536	1.1694	0.467	1.0263	0.527	1.32
0.358	0.8965	0.506	1.1828	0.412	1.0282	0.5861	1.316
0.371	0.845	0.526	1.0968	0.351	1.0708	0.6094	1.058
0.468	1.1221	0.48	1.09	0.366	1.0933	0.5274	1.176
0.478	0.9457	0.451	1.1791	0.41	1.0141	0.4766	0.995
0.406	0.8165	0.499	1.1796	0.428	1.1257	0.5112	1.302
0.5	0.8232	0.493	1.2077	0.361	1.018	0.4937	1.158
0.584	0.935	0.539	1.078	0.379	0.9961	0.5094	1.042
0.511	0.695	0.537	1.0106	0.419	0.8194	0.5115	1.13
0.533	0.6905	0.481	1.0588	0.293	0.7213	0.4385	0.892
0.705	0.628	0.481	0.9414	0.443	0.8657	0.4843	0.922
0.562	0.8161	0.486	0.9272	0.5	0.7737	0.497	1.005
0.807	0.5853	0.477	0.8803	0.453	0.665	0.5658	1.149
0.53	0.6224	0.535	0.881	0.405	0.8386	0.5986	0.922
0.621	0.6828	0.464	0.7683	0.378	0.7116	0.5798	0.723
0.5	0.6627	0.545	0.7868	0.307	0.635	0.5054	0.905
0.615	0.5701	0.591	0.7551	0.366	0.5416	0.3915	0.726
0.466	0.5182	0.516	0.776	0.377	0.6294	0.4359	0.705
0.472	0.5238	0.487	0.6399	0.434	0.6298	0.7204	0.792
0.426	0.4501	0.456	0.6464	0.33	0.4913	0.6209	0.625
0.453	0.4283	0.564	0.6762	0.389	0.5646	0.5424	0.703
0.643	0.4228	0.549	0.586	0.396	0.4238	0.4634	0.7
0.581	0.4306	0.579	0.6533	0.421	0.4551	0.3897	0.56
0.617	0.4027	0.547	0.5744	0.568	0.465	0.534	0.541
0.523	0.3339	0.467	0.6003	0.493	0.4706	0.5962	0.694
0.625	0.3563	0.434	0.5854	0.685	0.4449	0.6026	0.632
0.663	0.3632	0.529	0.4907	0.524	0.3809	0.5283	0.587
0.842	0.3531	0.545	0.5394	0.836	0.3894	0.5148	0.624
0.972	0.3791	0.544	0.5348	0.655	0.3724	0.4041	0.584
0.937	0.3717	0.592	0.4804	0.781	0.4295	0.526	0.484
0.71	0.4162	0.478	0.5871	0.894	0.4493	0.6446	0.653
0.911	0.3319	0.471	0.4916	0.775	0.4013	0.7309	0.555
0.977	0.3459	0.547	0.4907	0.989	0.4113	0.6194	0.491
1.03	0.3355	0.587	0.4777	1.026	0.3509	0.5398	0.468
0.956	0.385	0.533	0.4802	0.991	0.3994	0.4418	0.473
1.072	0.3342	0.491	0.4794	1.079	0.3877	0.5412	0.454
1.269	0.3545	0.481	0.4824	1.187	0.3803	0.5957	0.501
1.183	0.3405	0.52	0.5427	1.149	0.3592	0.5656	0.492
1.421	0.3454	0.517	0.4766	1.138	0.4311	0.5122	0.413
1.33	0.3687	0.597	0,4435	1.353	0.3973	0.5297	0.459
1.055	0.3741	0.545	0.5083	1.075	0.4286	0.486	0.555
1.169	0.3841	0.509	0.4394	1.534	0.4182	0.5499	0.605
1.277	0.3791	0.477	0.4505	1.544	0.4037	0.5794	0.528
1.037	0.3846	0.477	0.4189	1.244	0.4377	0.5278	0.46

MUSCULAR CHANGES DURING ONE YEAR OF FUNCTIONAL ELECTRICAL STIMULATION CYCLE TRAINING BY SPINAL CORD INJURED PEOPLE

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INTRODUCTION

Spinal Cord Injury (SCI) results in the complete or partial loss of sensory and motor function below the level of the lesion. This causes substantial muscular atrophy [1], slow to fast muscle fibre type transformation [2] and reduced blood flow [1] with severe reductions in muscle strength and fatigue resistance. Additionally, cardiovascular fitness decreases [3] and SCI people are vulnerable to cardiovascular diseases, type II diabetes and pressure sores. Functional electrical stimulation (FES) cycling [4] involves stimulation of lower limb muscles to elicit a cycling motion. Significant increases in muscle size [e.g. 5,6], fibre cross sectional area [7] and strength [e.g. 5,8] have been reported following FES cycling training. However, power output remains low (0-55 W). We investigated the effect of an intensive year long FES cycling programme on muscle properties of SCI people.

METHODS

Five SCI people (1 female) aged 45.2 ± 3.4 years (mean ± SEM), 173.04 ± 9.37 cm tall and body mass 71.88 ± 12.05 kg with complete SCI <T3 for at least two years were recruited. FES training was for one hour a day, 5 days a week for one year. Resistance was increased by a flywheel and gears. Subjects controlled stimulation intensity using a throttle and stimulation frequency was 50 Hz. Peak power output was measured at three monthly intervals by an incremental exercise test and fatigue during FES cycling was measured during 10 minutes cycling at 100 % stimulation intensity after 12 months. Magnetic resonance images (MRI) of the thigh, calf and gluteal muscles were taken at baseline and 12 months. Dynamometry was used to test for quadriceps maximal isometric strength and fatigue resistance [9] at three monthly intervals. Maximal voluntary force and fatigue resistance were also measured in 10 AB people aged 30.6 ± 3.2 years, 172.6 ± 1.9 cm tall and 69.5 ± 3.1 kg body mass.

RESULTS

Cross sectional area of the quadriceps, hamstrings, gluteals and gastrocnemius increased by 54.9 ± 9.5, 30.7 ± 3.4, 30.7 ± 13.7 and 31.9 ± 11.0 % respectively after 12 months training. Maximal torque significantly and progressively increased ($P < 0.05$) but remained significantly less than AB people ($P < 0.01$). Peak power output initially was 5.8 ± 1.0 W and improved significantly and progressively to 19.4 ± 4.5 W after 12 months. Relative changes in peak power output were substantially less than that of maximal torque (Fig. 1). Fatigue resistance during isometric contractions was significantly less than normal for SCI people and improved significantly ($P<0.01$) after one year to become more fatigue resistant than normal (Fig. 2). During 10 minutes FES cycling following the training programme there was a decrease in power output to 22.2 ± 2.4 % maximum after ~140 seconds. Thus SCI people were substantially less fatigue resistant during FES cycling than during isometric contractions of the quadriceps muscle (Fig. 2).

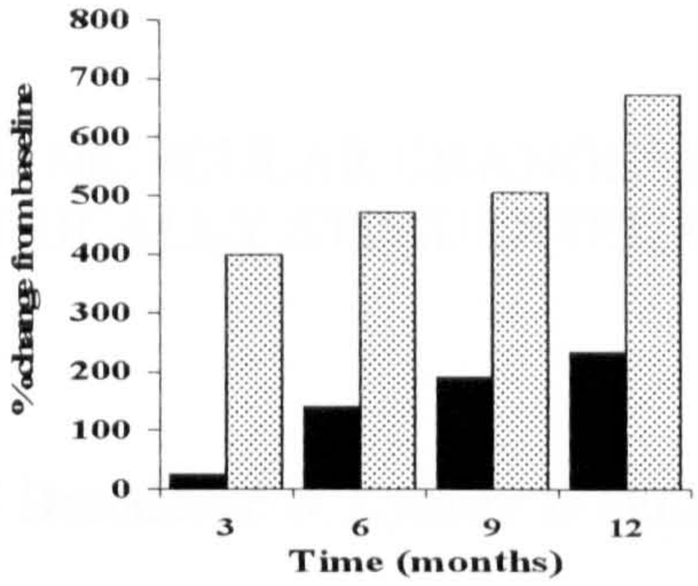

Fig. 1 Mean change of maximal quadriceps torque (grey bars) and peak power output during FES cycling (black bars) during the year of training.

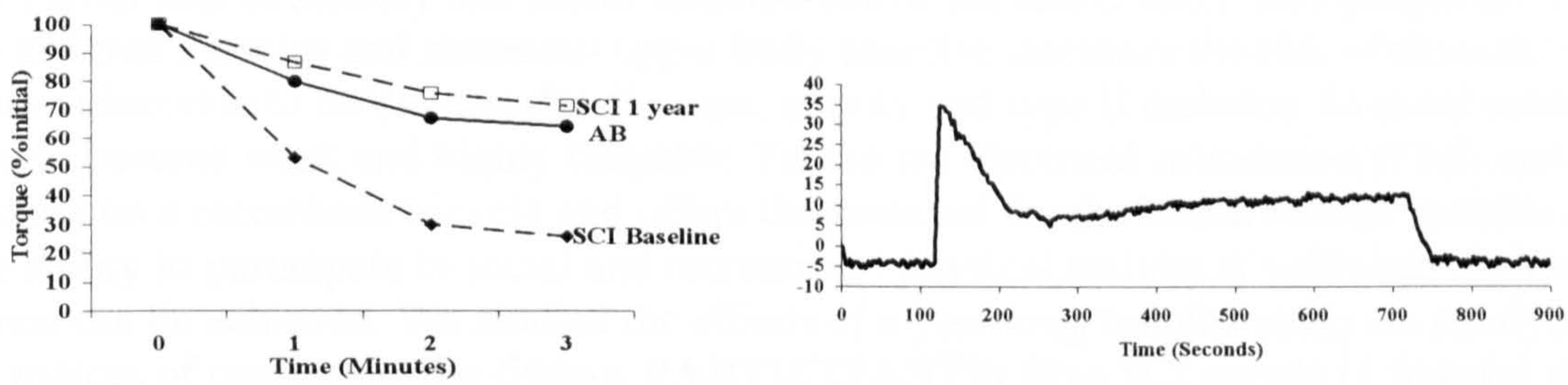

Fig. 2 a) Isometric quadriceps fatigue in SCI people (dashed line) before (closed diamonds) and after one year (open squares) of training and in AB people (solid line). b) Typical power output during 10 minutes FES cycling in one SCI person after one year of training.

DISCUSSION

Muscle size and strength and also peak power output during FES cycling improved significantly after training. The increase in power was much less than that of strength and remained generally insufficient for outdoor recreational cycling. This may be due to the timing and pattern of stimulation, the synchronous firing of motor units or the frequency used. There is an unexplained paradox in the high fatigue resistance seen after training during isometric quadriceps contractions and the rapid initial fall in power output during FES cycling.

CONCLUSION

While FES cycling training significantly increased strength and power there was a relatively small increase in power output. Power output declined markedly during cycling despite high fatigue resistance during isometric quadriceps contractions. Understanding the underlying mechanisms for these phenomena will enhance the recreational use of FES cycling.

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Appendix 18

TITLE
CARDIOVASCULAR AND MUSCULAR CHANGES DURING A ONE YEAR TRAINING PROGRAMME OF ELECTRICALLY STIMULATED CYCLING BY SPINAL CORD INJURED PEOPLE

AUTHORS
Di J Newham, Nick N de N Donaldson & Lynsey D Duffell

PURPOSE: To investigate the effect of an intensive year long FES cycling programme on cardiovascular fitness and muscle properties in SCI people. **RELEVANCE:** A spinal cord injury (SCI) results in complete or partial loss of sensory and motor function below the lesion level. SCI people are unable to activate the affected muscles and strenuous upper body exercise increases the risk of shoulder damage. Thus they are vulnerable to cardiovascular diseases, obesity and type II diabetes. Skeletal muscles below the lesion level become weak and highly fatigable. Functional electrical stimulation (FES) can be used to perform cycling on a recumbent tricycle and offers the potential for the health related benefits of exercise and also the ability to participate in social and recreational physical activity if sufficient muscle strength and endurance can be achieved. We studied the effects of a year long training study on paralysed skeletal muscle and indices of cardiovascular fitness. **PARTICIPANTS:** Five SCI people (1 female) mean (± SEM) age, height and weight 45.2 ± 3.4 years, 173.04 ± 9.37 cm and 71.88 ± 12.05 kg respectively, with complete SCI <T3 for at least two years were recruited with local ethical approval. **METHODS:** FES training was performed for one hour a day, 5 days a week for one year. Metabolic measurements were made during incremental and constant load exercise tests at three monthly intervals. Maximal isometric strength and fatigue resistance in the quadriceps were measured at three monthly intervals. **ANALYSIS:** ANOVAs were used to evaluate longitudinal changes. Where significant changes were identified, post hoc analysis was carried out using paired Students t-tests. **RESULTS:** Peak power output increased progressively from 5.8 ± 1.0 to 19.4 ± 4.5 W ($P<0.05$). Peak VO_2 improved by 42 and 66 % after 6 and 12 months, respectively ($P<0.01$). Steady state VO_2 at a constant work load reduced significantly ($P=0.01$) but HR during exercise remained unchanged. Quadriceps maximal strength significantly and progressively increased ($P<0.05$) as did quadriceps fatigue resistance ($P<0.01$). **CONCLUSIONS:** Although peak VO_2 increased significantly and to a greater extent than has been reported previously, it remained substantially lower than normal. Improvements in muscle strength and fatigue resistance probably allowed SCI people to attain a higher peak power output and therefore VO_2 after training. Reductions in steady state VO_2 probably occurred due to muscular adaptations e.g. an improved capacity for oxidative metabolism. Steady state HR did not change after training indicating that central cardiovascular improvements did not occur. The increase in muscle strength was significantly greater that that of power output ($P<0.05$). **IMPLICATIONS:** The cardiovascular responses indicate that the training programme should result in health related benefits for decreasing the risk of cardiovascular diseases in SCI people. Despite the improvements in muscle strength and fatigue resistance the power outputs achieved were sufficient only for cycling on a smooth flat surface. To optimise the functional and social benefits of electrically stimulated cycling for this population it is necessary to understand how power output can be enhanced.

KEYWORDS: spinal cord injury, electrical stimulation, health, cardiovascular fitness, skeletal muscle.

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